VOLUME 67 • APRIL 1958 • NUMBER 4

A.M.A. ARCHIVES OF

OTOLARYNGOLOGY

Office Management of Speech Problems
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Paul J. Moses

Anatomic Localization Implications of the Functional Hearing Tests
Scott N. Reger and C. M. Kos

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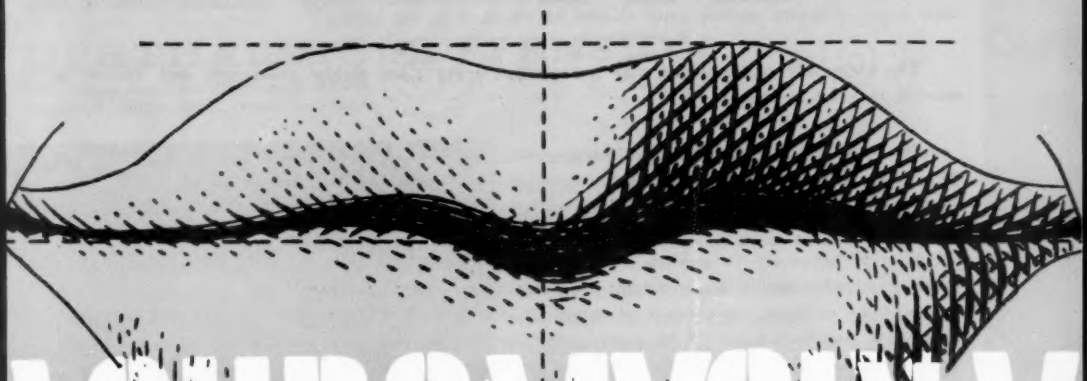

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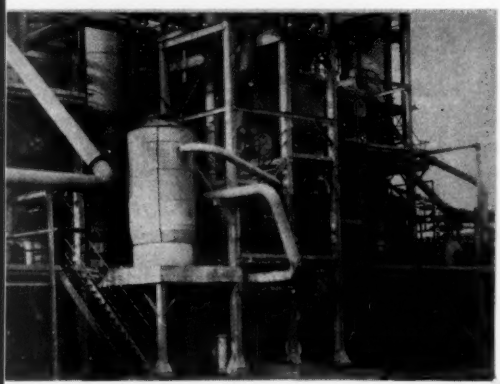

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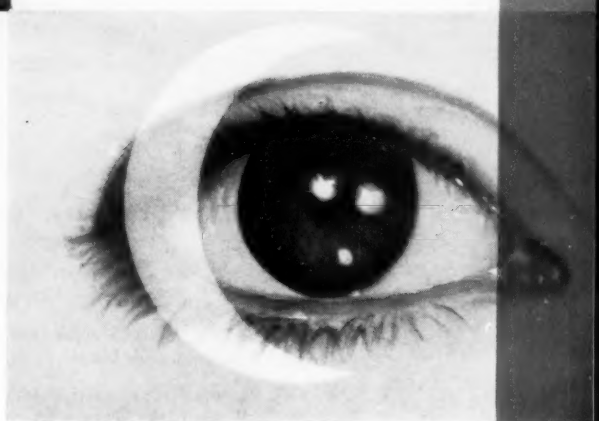

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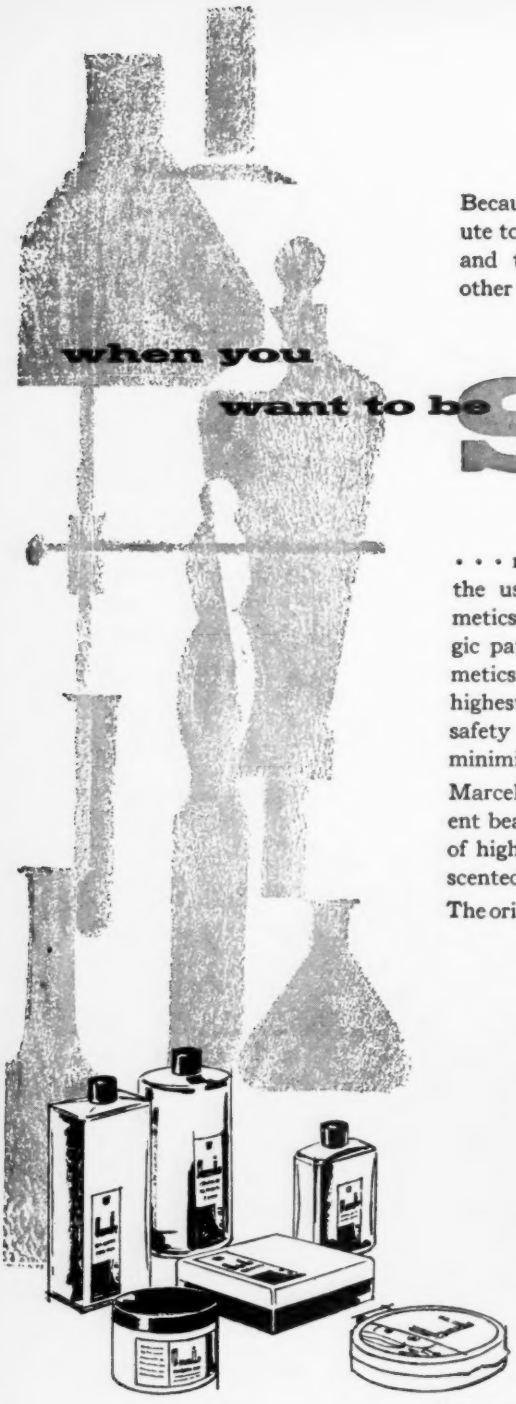

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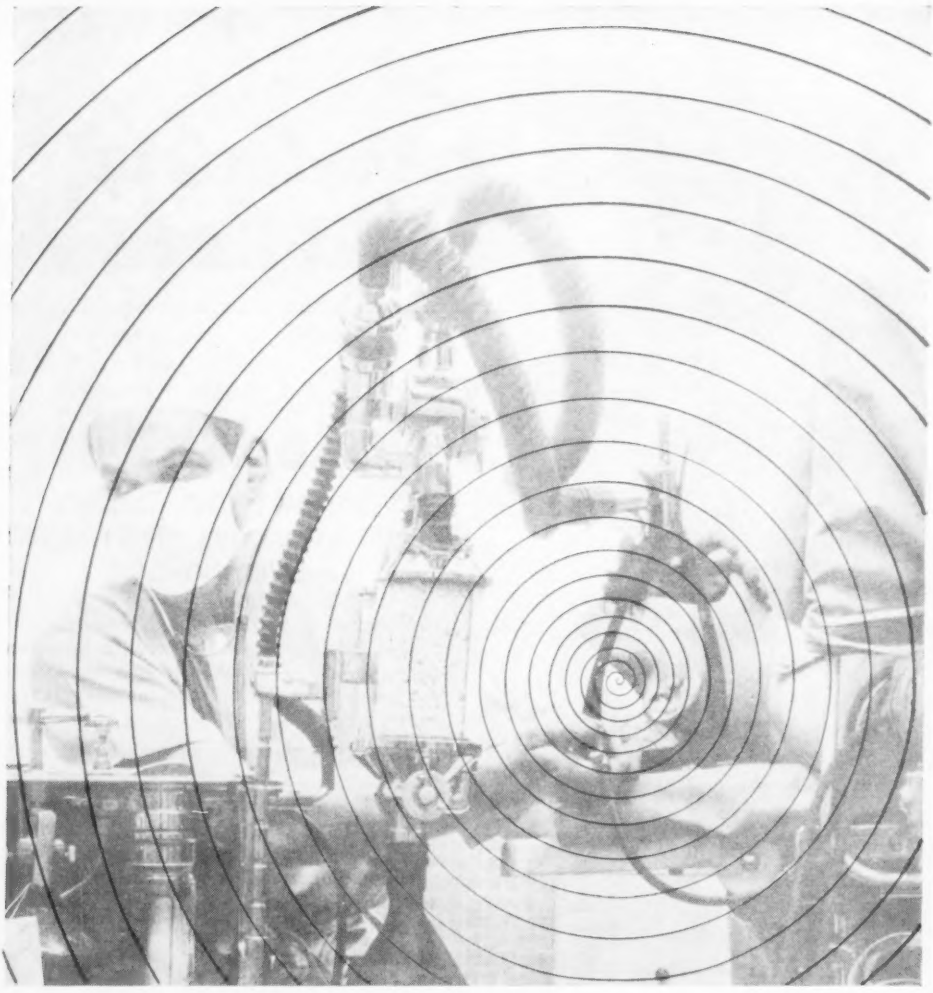

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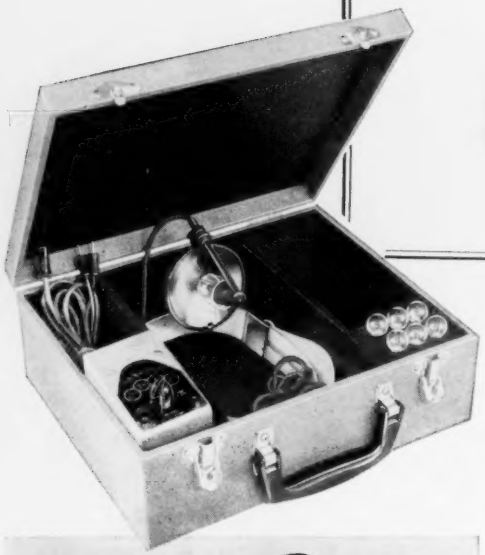

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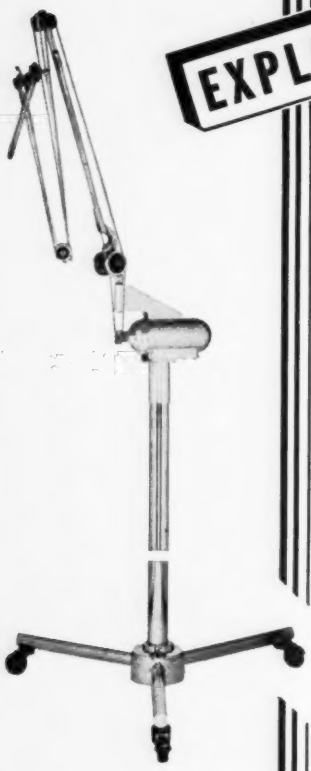

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Office Management of Speech Problems

HESTON L. WILSON, M.D., and WARREN R. DAWSON, Ph.D., Seattle

The majority of patients with speech problems can be handled quickly by the laryngologist in the office. He can, while pursuing the course of laryngeal pathology, institute vocal training as an adjunct to required medical or surgical treatment. Sometimes within as few as two or three visits the patient's speech rehabilitation is complete.

A close relationship between many speech problems and emotional disturbance has been conclusively demonstrated by Dr. Paul Moses.[1] Often the laryngologist can have speech rehabilitation well on its way while psychotherapy is being instituted.

The following cases will indicate how a simple, workable program for office management of speech problems is provided. The severer problems of speech, including stuttering, asphasia, and articulatory problems, are of course referred to a speech clinic or speech therapist.

The use of improper pitch is a very common type of vocal abuse. Froeschels[2] demonstrated clearly that faulty pitch produces uneven and inadequate closing of the vocal cords with resultant pathological changes in the cords themselves. Joel Pressman[3] in his monograph on the "Physiology of Vocal Cords in Phonation and Respiration" convincingly pointed out the effect of lowered pitch upon laryngeal structures.

The authoritative, low-pitched, "top-sergeant" type of voice is an example of faulty pitch. Its counterpart is the low, sultry female voice so frequently exemplified in the entertainment world. The high, thin voice in the male often is the result of failure to adapt to the pubertal growth of the larynx. In the female it is found in the tense, anxious patient.

In order to determine the proper pitch level of the patient, the following steps can be carried out quickly and easily in the office:

1. The patient is first asked to sing his lowest note. Frequently this is the pitch employed for his speaking voice. He is then asked to sing a scale. A very definite change in the resonance and timbre of the tone occurs about the fourth full tone above the lowest note that the patient is able to sing. A musically trained ear is not required to note the more pleasing quality of the voice at this level. The patient very often registers surprise at the ease with which these tones are sung.

2. The pitch is subsequently checked by having the patient place his fingers tightly in his ear canals and again sing the scale. He is asked to find the pitch at which his ears shake or vibrate the most. Usually this is quite apparent and the tone resolves itself at the level found by singing the scale.

3. Following the technique introduced by Froeschels the patient is asked to hum, at the same time making a vigorous chewing motion. He is told to make no attempt to hum a tune, but only to sing the note most comfortable to him.

The vigorous chewing prevents fixation of the mandible and hyoid, thus avoiding

Submitted for publication Aug. 5, 1957.

tension in the extralaryngeal musculature. The resultant tone is that of the patient's normal speaking voice.

The patient is then advised to use this as an exercise. He is cautioned against chewing gum or food while the exercise is being performed, and is asked to hum in a relaxed fashion, while keeping the jaw in motion. His ear therefore becomes accustomed to this tone and soon he adjusts to the normal speech level. He is admonished to do this exercise many times a day. It is not necessary that it be done for long periods. Only a few seconds are required to habituate the ear to the new speech level. He must however, make a concerted effort to maintain this speech level in normal speaking circumstances.

Diaphragmatic control in speech is another very common problem encountered in the office. This can be readily evaluated by asking the patient to count or recite a simple phrase while standing. Correctly, the thorax should remain stationary during expiration while the speaking breath is controlled by the diaphragm.

Two simple exercises can be employed to develop diaphragmatic control. In the first, the patient lies supine with a book placed upon the thorax, and another on the abdomen. He is told to breathe making the book upon the abdomen move, while that on the thorax remains stationary. As further control is gained, the patient is asked to practice speaking from the diaphragm before a mirror while disrobed, making certain that the thorax remains stationary. If possible, these exercises should be done for at least 15 minutes twice a day. Once the patient begins to achieve diaphragmatic control, he can practice while engaged in other activities.

The ensuing cases serve to illustrate the techniques and results to be expected:

CASE 1.—A 25-year-old white unmarried school teacher was first seen Oct. 7, 1955. She related that during her teaching career she had noticed severe hoarseness and pain about the neck and shoulders at the beginning of each school year. The hoarseness persisted throughout the school term, and the discomfort about the neck remained the same. During the summer months her complaints were relieved. The patient had taught for one month of this school term, and the hoarseness and laryngitis had become quite severe.

Examination was essentially negative, except for the larynx, which showed diffuse erythema with mild edema of the vocal cords. Analysis of the speech revealed no perceptible motion of the diaphragm; the air was being directed entirely from the upper thoracic cage. The patient was also using the lowest tones of her vocal range.

She was placed upon Froeschels' technique as described, and diaphragmatic exercises were begun. She was again seen Oct. 20. At this time the pitch was much improved; however, there was little progress in the diaphragmatic function. Evaluation revealed that the patient was misdirecting her breathing efforts, and with rediscussion of the exercise she returned again on Oct. 27 with an excellent result. Examination now showed that there was no erythema of the cords and the edema had decreased. She was highly pleased that her teaching ceased to be as strenuous as previously. The pitch of her voice had raised to its normal level, and the diaphragmatic action was well controlled.

CASE 2.—A 43-year-old white married woman was first seen May 3, 1955, complaining of marked hoarseness and soreness of her throat, which became worse toward the end of each day. She worked as a waitress where it was necessary to shout orders. Ear, nose, and throat history was otherwise negative. The physical examination was essentially negative. The larynx was found to appear within normal limits. Speech analysis revealed a voice about one octave higher than its normal pitch with accompanying harshness of quality and inadequate, shallow, upper chest breathing. The patient stated that she could speak with a lower tone. She deliberately avoided this, however, saying that it sounded too much like a man's voice. She was made to realize that her normal voice, even though at a low pitch, had all the characteristics of a female voice. With the institution of improved diaphragmatic control her improvement was rapid and the hoarseness and discomfort were readily relieved.

CASE 3.—On June 20, 1955, a 46-year-old white woman gave a history of hoarseness since the previous November. There was considerable soreness of the throat which had persisted from the outset. The present illness was associated with considerable marital discord. When she was first seen, marked anxiety was exhibited. Examination showed no significant ENT findings other than injection of the true vocal cords. Speech analysis revealed the speech level established at a very low pitch. Respirations with speech were highly in-

adequate, and she resisted psychiatric evaluation. Diaphragmatic exercises and the Froeschels' technique were instituted. She was again seen on June 27 and displayed good speech function, but there was still some difficulty with the diaphragmatic control. Psychiatric consultation was obtained on July 11, and with psychotherapy in addition to her speech therapy her voice readily returned to normal within a six-week period.

CASE 4.—A 43-year-old white man was observed Feb. 6, 1956. Severe hoarseness had been present for eight weeks. Some nonproductive cough had been present.

Examination revealed a large polyp present in the middle one-third of the left vocal cord. On Feb. 12 the polyp was removed by direct laryngoscopy. Voice rest was maintained for one week.

Analysis of the patient's speech had revealed good diaphragmatic control; Froechels' exercises were begun, and when the patient was seen on Feb. 28 the voice had returned to normal pitch.

The patient was again examined in May, 1956. The larynx appeared within normal limits. Speech was found to be markedly improved.

In summary, a simple, workable method of speech therapy applicable to office practice has been outlined. This takes little additional time over and above the time employed for the usual office visit. The four characteristic cases presented demonstrate the efficacy of this system.

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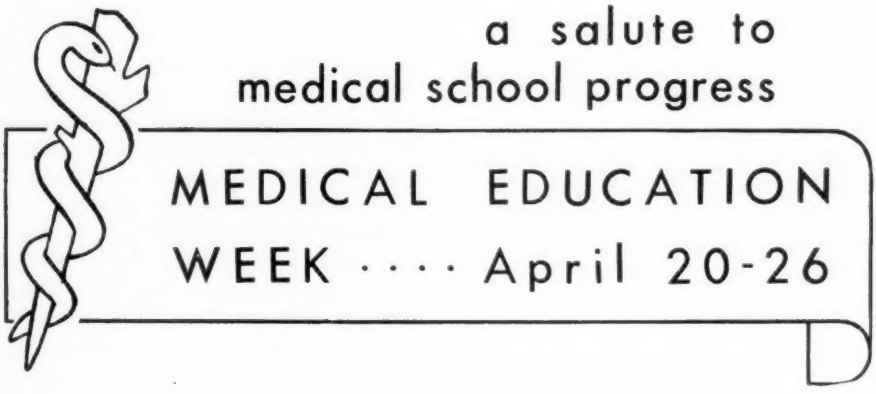

Psychosomatic Aspects of Inspiratory Voice

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Observations on inspiratory voice are rarely made in the literature on phonetics. The few published case histories describe the physiological facts and processes by which this vocal phenomenon is produced—contrary to normal phonation—on inspiration instead of expiration. As therapy the authors all give the obvious advice to retrain the patient from inspiratory phonation to an expiratory one. None of these reports pay attention to the personality of the patient who had consulted these "fact-finders" and who usually resisted their therapeutic efforts.

In previous publications I have pointed out how every transitional step between phonation on complete expiration and on complete inhalation can be used as a neurotic expression.[1] Most frequently one finds under these conditions a type of exhalation which keeps the intercostal muscles in an inspiratory position. The result is often a quavering voice produced through tonus changes within the vocal cords. In vowel stuttering in children this tonus can reach such a spastic character that only inspiratory pressure can release it; thereupon the stutterer starts talking on inspiration.[2]

A complete inspiratory voice sounds as if the tones are sucked in. The acoustic diagnosis cannot be missed. Instinctively one would suppose that shifting the voice from inspiratory to expiratory phonation would be all that is necessary. The poor therapeutic results from this method indicate that we face a much more complicated and involved problem, because even when the symptom can be temporarily improved, the causal factors of the neurosis resist treatment and the symptoms often reappear in some other form. The profound symbolic meaning of this symptom can be detected in anthropological, archaic-mythical, and linguistic sources. It can be traced as an utterance which belongs to the "forgotten language" of dreams, myths, fairy tales, and ancient symbols.

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Brief Review of Inspiratory Sounds

Although in our Western languages this symptom of inspiratory voice makes communication unsuccessful and therefore belongs to disturbed expressive behavior, in South Africa the Hottentot and Bushman languages have also a number of inspiratory sounds, pronounced by sucking in the breath at various points of oral contact, the so-called clicks.[3] These differ from all other phenomena of speech by creating a vacuum in the mouth into which air is introduced, making a sharp sound.[4] When lips are used, a kissing-like sound results, and this may point to a possibly sexual origin of language. Vowel sounds may have originated from sex calls. Sucking and breathing sounds share a sexual and ingestive significance. The English words *suck, sip, soup, sup* signify the inspiratory function. The pronunciation of the letter *l* includes inspiration and the touching of mucous membranes, plus production of saliva. Interest in sex that the law forbids is expressed in words beginning with an inspiratory *l*: *lewd, lascivious, lecherous, libidinous, lustful.*[5] Good food is called by words with the sucking *l*, as in *delicious, delightful, delectable*. Thus, the inspiratory voice is used to express joy, well-being, good food, good drinking, and sex.[6]

As vocal gesture, the inspiratory "brrr" is used to hold back the horses in pulling the reins. One makes implosive sounds by inhalation in "tststs" to express sympathy. One sucks in "FFF" in pain, and "oh" in unbelieving horror and in indignation. This is somatic symbolism of certain vowels and consonants, the sound image of holding something between the lips, to express closing, containing, gripping action. Inspiratory sounds produced when one swallows good food mean and emphasize the effort to retain the good taste.

Connection of Inspiratory Voice with Feeding

Since the larynx was originally not a phonatory but a respiratory organ, its sphincter action can easily be compared to the ingestive organ. Air intake has its parallel in food intake. Breathing is a form of feeding. Sounds, too, are produced in eating, drinking, and breathing. Speech becomes a by-product of air hunger and food hunger. Voice eventually forms itself through its relation to the external world, to which it appeals in an ambivalent way.

The meaning of ingestion, of incorporation, of holding back becomes evident. Sucking and the using of inspiratory voice become intimately related very early, during the phase of life when the child communicates with the mother through sucking. Later inspiratory voice can become the expression of a repressed wish for the mother. Hunger for sucking can recur in patients of any age, if this mode of communication through mother's breast was denied in childhood.[7] Asthma of psychogenic origin has been explained as a cry for the help of the mother, whom the patient tries to introject by respiration so as to be permanently protected.[8]

Since smelling and sniffling in infancy are connected with sucking, they also become a source of pleasure. In later neuroses these former pleasures can be remobilized, and the intake and outlet of air may then symbolize incorporation.[9] In primitive thinking this inhalation of air plays an important part. Inhaling the same air as another person means to be united with him, which is even close to our present thinking. In many primitive tribes the last breath of the dying chief has to be inhaled by his successor, and it can be caught in a bag. It contains the soul. Supernatural powers can be acquired through inspiration. For instance, oracles are made possible only when the priest sucks the blood of a sacrificed victim. The priest in Uganda smokes a special pipe, inhaling and speaking with the voice of the god. In other tribes the priestess inhales smoke from special kindling fire and the spirit enters her.[10] In all these cases the inhalation has the symbolic meaning of incorporation. In certain parts of England "eating the sop" belongs to the wedding ceremony; bride and bridegroom suck from a morsel dunked in fat in order to be united, like breathing the same air.[11] The word "sop" expresses the inspiratory character in an onomatopoetic way.

Whereas inspiration means unification, exhalation has the hidden dangerous significance of separation. Inspiratory voice can therefore be a cry for help, a cry for mother, as before described in asthma.

Respiration and Anxiety

The intimate connection between anxiety and respiration is well known. Every variation of respiratory function expresses a certain degree of anxiety. Major changes of breathing occur in more intense forms of anxiety. These changes can be used as equivalents for anxiety; for example, changes in body functions, in somatic sensations replace affects.[12] These affects are then reduced to or translated into the language of the body. Primitive people, psychotics, and children express this type of translation of affects more clearly and more openly. The neurotic reveals through inspiratory voice that intake and outlet of air can symbolize incorporation and projection of what had been incorporated. It therefore fulfills a function, for instance, to keep the protecting person, to remain protected, while

projection through exhalation would mean to renounce this protection.

The symptom in itself influences the external world. The patient gains advantage through pity. He can constantly express his increased demands on his surroundings, such as demands for attention and love, advantages which he will not give up easily. In several cases which I described previously the symptom meant a secret which the patient tried to keep, to retain. This urge for retention was translated into body language by speaking on inhalation.

In these cases the content of the secret made it understandable that the factor of guilt was also involved, together with the subsequent need for self-punishment. The inspiratory voice is produced with tremendous effort. Not one tone is produced easily. The self-punishment consists in this effort, which can be described as a hyperfunction. This term is meaningful only if the psychogenetic factors are described which cause it.

Hyperfunctional effort eventually always results in visible organic irritation of vocal cords. Potentially the patient is well able to talk on expiration, and to use it would make talking so much easier for him. When he sometimes "slips" and uses normal voice, he corrects this immediately as if he has done something wrong, and again starts "sucking it in." Something stronger than the painful effort is at work and forces him into this punishment. It is punishment inflicted upon oneself to undo guilt feelings. The secret which should not be told or known is the motive behind it—in one case a son born out of wedlock, in another a love affair and marriage which broke up a family.

Characteristic is the story of a missionary physician who had deserted his flock among the South American Indians and had returned to the United States only to move restlessly from one location to another, with increasing inspiratory and persistent falsetto voice. In his case combination of the two vocal mechanisms expressed the desire for mother and revealed a still unsolved Oedipus situation, associated with guilt feeling and need for self-punishment.

Inspiratory voice is often observed in certain parochial schools in the function of whispering; here silence means submission, and talking is often discouraged even during recess periods. Teen-age girls are also likely to use this type of whispering in which the suppressed sexual character of the symptom can be detected.

Therapy

Critics have often warned against the overpsychologizing of vocal phenomena. These warners should stop to think that they may be on the alert, afraid something might be found out about them. In their writings these warners have always tried to impress the reader with a kind of therapy which evades the necessity for unveiling the psychomotor forces behind the symptom. Inspiratory voice needs a special therapy. Here descriptive phonetics have to be changed into an interpretative process. We face a symptom of ambivalent tendencies, of opposite attitudes, of wanted and at the same time inhibited communication, of expression, suppression, and repression.[13] The patient holds back, retains, and will be cured only if he has an opportunity to express himself fully—not just vocally: if he comes to understand some of his difficulties. Types of shock-like procedures, of surprise attacks, are still recommended by authors without insight into psychodynamics and used, for instance, in aphonia and phonasthenia, such as touching the vocal cords with metal balls ("Muck'sche Kugel") or faradization.[14] Any of these will do nothing but make the patient transfer his symptom to another organ. Understanding of the psychodynamics of inspiratory voice should contribute to a reorientation in understanding and treating functional voice disorders.

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Anatomic Localization Implications of the Functional Hearing Tests

Revision of Terminology

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Synopsis: This paper is concerned primarily with an evaluation of the terminology commonly used to express the results of the functional hearing tests. At the present time the nomenclature lacks a unifying logical coherence, a lack which often results in vague generalities and loose thinking about certain ear pathologies and diagnoses. For example, the phrase "middle ear" involvement has an anatomic connotation; "conduction" impairment conveys a physiologic implication, and "perception" deafness suggests psychologic implications—three different classification schemata or frameworks. We expound the thesis that a cohesive system of terminology based solely on the anatomic localization of pathology associated with hearing impairments is preferable from the diagnostic and treatment points of view.

I. Diagnostic Role of the Functional Hearing Tests

Unequivocal recognition of the diagnostic capabilities and limitations of the functional hearing tests precedes rational interpretation of the test results. The fact that the data obtained by means of the tests do not constitute the diagnosis but serve as diagnostic clues only cannot be emphasized too forcibly. It is axiomatic that otologic diagnosis be based on careful consideration of the history, physical examination, and the results of the hearing tests. Since direct physical examination of lesions medial to the middle ear usually is impossible, studies of the pathophysiology of the auditory mechanism have been found invaluable in the differential diagnosis of hearing disorders. By means of various types of functional hearing tests it is possible to penetrate and explore in anatomic regions within the hearing mechanism where the eye cannot follow. Fortunately, the various anatomic structures which participate in the hearing process perform highly specialized functions, many of which can be isolated, measured, and evaluated in terms of the test results. The tests aid in formulating the diagnosis by indicating the anatomic localizations of the pathophysiology responsible for the hearing abnormality. Treatment, surgical or otherwise, is concentrated either on anatomic structures or on the agents responsible for the pathologic involvement in question.

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II. Inadequacies of Older Terminology

With the considerable emphasis on the diagnostic significance of altered auditory physiology, it is not surprising that the results of the tests often are expressed in physiologic rather than anatomic terms. And since most of the measurements of the integrity of the hearing process are based on the subjective responses of the patient, it is equally obvious why psychologic terminology also has been employed to designate certain hearing aberrations. These etymologic factors suggest the origin of the anatomic, the physiologic, and the psychologic classification schemata that have been used as diagnostic clues or aids.

We are convinced that adherence to a single classification schema, based on ana-

tomic localization of pathology, is preferable from the diagnostic point of view. Tests of physiologic behavior of the auditory system by means of the functional hearing tests are but a means to an end—identification of the altered structure (or structures) responsible for the hearing abnormality. Anatomic localization and identity of pathology necessarily precede diagnosis and treatment.

It has been common practice to divide ear involvement (ear pathologies or hearing impairments) into (1) middle ear and (2) nerve or perceptive types. (The simultaneous presence of both types has been called mixed involvement.) The inadequacies of this simple dichotomous classification were emphasized in 1948 by Dix, Hallpike, and Hood,[7] who pointed out that a distinction between at least two different types of *"not* middle ear" deafness could be made by means of recruitment tests.

At the present time the terms "middle ear" deafness, "tympanic" deafness, "conduction" deafness, "transmission" deafness, and "impedance" deafness often are used interchangeably by different writers; also the terms "nerve" deafness, "perception" deafness, "acoustic neuritis," and "inner ear" deafness often are used in an indiscriminate interchangeable manner.

III. Preferred Terminology

By way of introduction to the proposed terminology it is suggested that ear involvements be classified as (1) tympanic, (2) cochlear, and (3) neural. This anatomic localization classification is not entirely new. However, it is believed that adherence to this simple terminology with its logical implications will induce the examiner to be highly critical of the results of his functional hearing tests, thereby enabling him to make a more accurate diagnosis. The term "tympanic" is synonymous with "middle ear" but is preferred because of the convenience with which prefixes can be used with the shorter term. For example, it is obvious that the term "laterotympanic" refers to pathologies lateral to the tympanum, such as atresias of the external auditory canal; the term "mediotympanic" also is useful when referring to pathologies medial to the tympanum. ("Mediotympanic" is preferred to "retrotympanic," since "medio-" means "nearer the median plane" and "retro-" implies "behind.")

The terms "conduction" deafness, "transmission" deafness, and "impedance" deafness do possess certain tympanic localization implications since each usually includes or involves the tympanic membrane and the auditory ossicles. However, the tympanic localization aspects of these terms has been complicated and obscured by occasional references in the literature to "conduction deafness of the inner ear" and "impedance deafness of the inner ear." The above three terms also convey significant physiologic inferences since it is the function of the tympanic membrane and auditory ossicles to "conduct" or "transmit" vibrational energy from the atmosphere to the inner ear fluids.

It is believed that maximum simplification and clarification of nomenclature will result if the term "tympanic" pathology (or tympanic deafness or tympanic involvement) be used to denote the localization of ear pathologies which involve the tympanic structures. Ordinarily, tympanic pathology cannot be localized in any one of the several vibratory structures associated with the tympanum by means of the functional tests alone. However, if by means of the combined history, physical examination, and hearing tests, it can be demonstrated that the significant pathology is confined to a single anatomic entity, such as the stapes, this conclusion could be summarized as "stapedial involvement" should the examiner prefer.

The hearing test results are somewhat similar in practically all types of tympanic (or laterotympanic) diseases or conditions, such as atresia of the external auditory canal, otitis media, and otosclerosis—to name but a few possibilities. The three named diagnoses cannot be differentiated accurately on the basis of the hearing tests

alone, but only after consideration of the history and physical examination. It is possible that the otologic diagnosis in turn may suggest a more exact or circumscribed location of the pathology within the tympanum than was possible by means of the hearing tests. For example, the diagnosis of otosclerosis not only implies involvement of the footplate of the stapes, but in addition indicates a body of knowledge about the etiology, treatment, and prognosis of the disease. As is obvious, considerably more needs to be known about the patient to make a diagnosis of otosclerosis than the results of the hearing tests.

As indicated above, it has been common practice in the past to dichotomize ear pathologies into : (1) tympanic and (2) nerve or perceptive types. The terms "nerve" and "perceptive" often are used synonymously and interchangeably to denote "not-tympanic" involvement, and imply that the locus and nature of the pathology are unknown except when the history and physical examination indicate a specific otologic diagnosis, such as a tumor of the eighth nerve. This latter usage of the term "nerve," since it possesses significant anatomic localization implications, does belong in the suggested nomenclature. However, the term "perception deafness" does *not*, since it is not an anatomical term, and its localization implications are relatively nonspecific. So-called perception deafness may result from pathologic involvement of the hair cells of Corti's organ, the eighth nerve, higher auditory nerve tracts, or the auditory cortex, including the association centers. No objection is raised to the use of the term "perception" as an indication of the ability of the patient to understand or perceive audible speech. It seems more appropriate to regard perception deafness as a symptom rather than a disease entity.

The term "cochlear," as used herein, refers to the nonneural contents of the cochlea. Here again we prefer the single word "cochlear" to such terms as "end organ" or "inner ear" because of the convenience with which the prefixes "latero-" and "medio-" combine with "cochlear."

The division of hearing impairments into tympanic, cochlear, and neural suggests a certain amount of confidence in the ability to demonstrate these distinctions—an assumption not always confirmed by clinical experience. For example, the examiner may be able to rule out tympanic involvement on the basis of the ear history, physical examination, and hearing tests, but be unable to localize the pathology in either the cochlea or in the neural pathways, the one to the exclusion of the other. Until the localization of the pathology can be ascertained with a high degree of confidence it is convenient (and truthfully realistic) to refer to such a condition as "mediotympanic"—implying that the pathology exists somewhere between the tympanic structures and the midline. It is obvious that the term "mediotympanic" is more or less synonymous with the older terms "perception" and "nerve." But labeling a hearing impairment "nerve" deafness simply because tympanic pathology is not demonstrable is misleading unless involvement of neural tissue can be confirmed.

Instead of subdividing ear involvements into (1) tympanic, (2) cochlear and (3) neural, it may be expedient at times to classify the anatomic localization of ear pathologies as follows:

I. Tympanic
II. Mediotympanic
    A. Cochlear
    B. Neural

Usually, tympanic involvement can be confirmed or ruled out with a high degree of certainty. The differentiation between cochlear and neural impairments may be exceedingly difficult and require considerable time and study. Unless a "not-tympanic" type of pathology can be localized with demonstrable accuracy, it is convenient to refer to the involvement as mediotympanic until additional discriminatory evidence is obtained. The term mediotympanic is merely a "stopgap" until a diagnostically useful localization of the pathology can be made.

IV. Localization Criteria

A. Tympanic Involvement.—As early as 1589 Hieronymus Capivacci [6] divided hearing impairments into those resulting from diseases of the tympanic membrane and those resulting from diseases of the labyrinth. His testing armentarium consisted chiefly of a musical instrument—the zither. (The tuning fork was invented by John Shore in 1711.) If the patient could not hear music played in the conventional manner on the zither, Capivacci placed one end of a wooden rod between the teeth of the patient and the opposite end in contact with the body or lower end of a string of the instrument. If the patient heard the tone through the rod when the string was plucked, the condition was diagnosed as a disease of the drum membrane; if he did not hear the tone, as a disease of the labyrinth. Capivacci's classification of hearing involvements has endured up to the present time, and his use and interpretation of bone-conduction sensitivity testing remains one of the most useful functional hearing tests in use today.

In spite of much adverse criticism directed against BC sensitivity tests, several investigators have demonstrated that the accuracy of BC tests rivals that of the AC tests if given under carefully controlled conditions. In general, patients with hearing impairments for AC sound but normal hearing for BC sound have pathologic involvement of one or more tympanic structures. In theory, if both the oval and round windows were to become completely immobilized, the vibration of the cochlear fluids would be greatly attenuated, resulting in an equal decrease for both AC and BC sensitivity. Fortunately, such cases are extremely rare. Patients with normal BC do not have AC losses in excess of 60 db.; the AC sensitivity curve shows approximately equal db. losses over the audiometric range; and speech discrimination remains essentially normal if the speech is presented at a sufficiently high intensity level. Mobility tests, such as the Gellé and Bing, as well as impedance measurements, show that the mobility of the ossicular chain is markedly reduced. Tympanic deafness results from any pathology which interferes with the mechanical vibratory efficiency of the tympanic membrane and ossicular chain.

B. Cochlear Involvement.—Certain test results are identical in both cochlear and neural pathologies. Both show approximately equal db. losses for AC and BC sound; the tympanic membrane and ossicles retain normal mobility (sensitivity to vibration), with the possible exception of patients with markedly increased intralabyrinthine fluid pressure (labyrinthine hydrops); and speech discrimination usually is seriously impaired, regardless of the intensity level at which the speech is presented. As a matter of fact, most cochlear and neural pathologies exhibit more similarities than differences.

As briefly mentioned earlier, Dix, Hallpike, and Hood [7] in 1948 reported a study which concluded that recruitment was experienced by patients with labyrinthine hydrops but not by patients with hearing impairments resulting from acoustic neurinomas. (The perceptual phenomenon which is now called recruitment was described by Pohlman and Kranz [13] in 1924.) Previous to 1948 it had been assumed generally that patients with "nerve" or "perception" deafness experienced the recruitment symptom but that those with tympanic deafness did not. Recruitment tests were performed to help differentiate tympanic deafness from nerve deafness. However, so many localization clues were already available with which to demonstrate the absence or existence of tympanic pathology that relatively little interest was aroused about recruitment tests so long as the dichotomous division of ear involvement was accepted.

The article by Dix, Hallpike, and Hood emphasized the inadequacies of the dual "conductive deafness—perceptive deafness" categories and accelerated renewed interest in recruitment testing. Recruitment tests now appeared to provide a technique by

means of which two different types of mediotympanic pathologies could be identified and localized to more specific anatomic regions.

It has been learned that persons with temporary or permanent hearing losses resulting from noise exposure also experience recruitment. This type of loss is known to involve the hair cells of the organ of Corti. (The term "hair-cell" pathology may be preferred to cochlear pathology in this instance.)

Many investigators refer to recruitment as if it were a pathologic entity possessing physical characteristics which exist in an anatomic site which can be seen under a microscope. Recruitment is a perceptual phenomenon—a psychological concept. Instead of using the common phrase "the patient has recruitment" it is more correct to say "the patient experiences the recruitment symptom." (As the result of additional correlation of hair-cell or other pathology with symptomatology, it may be possible in the future to observe the pathology in question and predict the probability that a given temporal bone donor had experienced the recruitment symptom.) It may be that certain pathologies of cochlear structures responsible for hearing impairments do not give origin to the recruitment symptom. It may be that a given cochlear disease—labyrinthine hydrops, for example—will induce the recruitment symptom in its early stages but not in the later stages. The presence of the recruitment symptom in patients with hearing impairments resulting from acoustic nerve tumors has been reported since publication of the informative study by Dix, Hallpike, and Hood.[7] It may be that the recruitment symptom associated with certain eighth nerve tumor cases is a residuum from previous cochlear pathology, such as that which results from high intensity level noise exposure.

The recruitment symptom presents many complicated facets which need additional unbiased investigation from the standpoints of psychophysical characteristics, validity of test techniques, quantification of measurement, etiology, interpretation of measurement, and so on. At the present time it appears that the recruitment symptom is associated with certain types of cochlear pathologies and may not be characteristic of uncomplicated neural involvements.

Pitch perception aberrations have been noted in certain types of ear conditions for several decades. For example, it has been observed that patients with labyrinthine hydrops usually perceive a given frequency as higher in pitch in the involved than in the normal ear. This discrepancy has been called diplacusis binauralis dysharmonica. Shifts in pitch also have been reported after exposing the normal ear to sounds possessing the dimensions required to decrease the sensitivity of the ear a certain amount. It appears significant that at least two cochlear pathologies—labyrinthine hydrops and acoustic trauma—which are associated with pitch aberrations also are associated with the recruitment symptom.

The pitch discrimination ability (difference limen for frequency—DLF) of the pathologic ear has received attention during the past few years by several investigators, including Shutts,[17] Schubert,[15] Azzi,[1] Fournier,[10] Meurman,[12] Filling,[9] and Butler and Albrite.[5] These early studies have made valuable contributions to the differential diagnosis of ear pathologies. Most of the authors were influenced strongly by the dichotomous "conductive-perceptive" division of ear pathologies and regarded the DLF measurement as a technique by means of which tympanic involvement could be differentiated from mediotympanic involvement—as was the case with the recruitment system previous to Dix, Hallpike, and Hood. However, as stated earlier, adequate and simpler tests exist for distinguishing between tympanic and mediotympanic impairments. Proof that patients with cochlear pathology possess worse (or better) pitch discrimination than those with neural pathology would be of greater value.

It appears plausible that pitch discrimination may be impaired as the result of certain types of cochlear pathology. It may be that recruitment and poor pitch discrimination are closely associated, since pathologic involvement of the hair cells appears to be related in both the recruitment symptom and reduced pitch discrimination. Additional studies which correlate quantified recruitment, the DLF, speech discrimination, and temporary threshold shifts (TTS) resulting from near threshold intensity level exposures are needed to learn the specific significance of the DLF in the pathologic ear.

C. Neural Involvements.—The localization of lesions at various neurologic levels within the auditory pathways often presents baffling problems. If the results of the ear history, physical examination, and functional hearing tests are not decisive, the vestibular tests, EEG studies, and a more complete neurologic examination may supply the determining localization or diagnostic clue. However, as a result of the relatively recent intense interest in speech audiometry, PGSR audiometry, and adaptation or temporary threshold shift (TTS) measurement, rapid strides (if new ideas can be considered such) are being made in the localization of neural pathologies by means of the functional hearing tests.

The writers are finding the measurement of TTS for threshold intensities by means of a Békésy-type audiometer an exceedingly reliable localization clue in the diagnosis of eighth nerve tumors. This observation on a single case was reported by Reger and Kos [14] in 1952. Since that date six additional cases of eighth nerve tumors (all verified by surgery) have been seen by us; all have demonstrated the development of abnormally rapid TTS's for barely audible tones.

A few months after publication of the above article an intriguing short paper by Gradenigo,[11] published in 1893, "On the Clinical Signs of the Affections of the Auditory Nerve" attracted our attention. Gradenigo had the following to say about variations in threshold sensitivity resulting from exposure to test tone stimulation: "Another important clinical characteristic with which I have met in some affections of the acusticus, consists in an excessive functional exhaustibility. According to my experience, this symptom may be met with, although very seldom, in middle-ear affections, especially in neurasthenic individuals. This exhaustibility is also often produced by alterations in the inner ear, but only in affections of the auditory nerve does it reach a sufficiently marked degree to be characteristic. The functional exhaustibility is often so considerable that the results of the examinations by the different methods become so essentially modified that it is impossible, if no account is taken of this fact, to form an accurate conclusion as to the amount of hearing power present."

Additional clinically useful progress has been made during the present decade in the localization of neural pathologies. In 1953 Bordley and Haskins [4] reported the results of PGSR audiometry on a group of older patients who could not obtain satisfactory results with hearing aids. "It would seem, then, that in some instances the conditioned reflex type testing as employed in psychogalvanic skin-resistance audiometry measures only the efficiency of the auditory end organ and the sub-cortical auditory pathways, and that measurements by subjective audiometry represent the performance of the hearing mechanism as a whole from the external ear through the auditory cortex. If such is the case it would seem reasonable to think that those individuals with good hearing by objective audiometry but poor hearing by subjective audiometry, with poor discrimination performance, and inability to use amplification suffer fundamentally from damage to the cerebrum, probably to the cerebral cortex. . . . The clinical and laboratory observations, considered together, suggest that the difference between the hearing thresholds by subjective and by objective audiometry indicates approximately the degree of cerebral sclerosis of the auditory area."

After reviewing the pertinent literature Bordley [4] concluded: "It would seem then, that the auditory-sympathetic reflex must lie somewhere below the cortex and above the level of the inferior colliculi and the rostral border of the pons. . . . PGSR audiometry would seem to measure only the efficiency of the auditory end-organ and the subcortical auditory pathways, while standard audiometry measures the efficiency of the hearing mechanism as a whole from the external ear through the auditory cortex. It evaluates the reception rather than the perception of sounds."

Bocca, Calearo, Cassinari, and Migliavacca [2] published a provocative study on "Testing 'Cortical' Hearing in Temporal Lobe Tumors," presenting a technique which showed significant differences in speech perception between the healthy and the involved hemisphere under certain test conditions. Each of their 18 patients who had supratentorial cerebral tumors had approximately bilaterally equal threshold sensitivity for pure tones and essentially identical bilateral preoperative articulation curves for normal voice. When the Bocca and Pellegrini disyllabic meaningful word lists were presented to each ear separately through a low pass filter which had an attenuation characteristic of about 30 db. per octave above approximately 400 cps (40 db. attenuation at 1000 cps), the articulation curves showed significantly lower scores in the ear contralateral to the cortical lesion. "The impairment of the articulation curve for the distorted voice disappears often some time after the surgical removal of the affected temporal cortex, as an effect of compensation well known in other fields of neuropathology."

The results of the Bordley and Haskins [4] and the Bocca et al.[2] studies suggest the possibility that a combination of conventional pure-tone audiometry, PGSR audiometry, and speech audiometry (complicated by noise, frequency limitation, and speed of delivery) may be helpful in differentiating between neural pathologies at the cortical and the subcortical levels.

The localization implications of tests of speech sound discrimination or intelligibility must be interpreted with extreme caution. It is common knowledge that patients with tympanic impairments usually understand amplified speech as well as persons with normal hearing if the intensity of the speech is sufficiently great. It is also generally accepted that patients with recruitment usually experience disproportionate difficulty perceiving (understanding) speech, regardless of the intensity levels at which the speech is presented. As a matter of fact, cases with marked impairments and severe recruitment may experience a reduction in speech intelligibility if the intensity of the speech is increased above a certain optimum value.

The value of the speech tests in relation to the diagnosis of eighth nerve tumors is not incontrovertibly established at the present time. Dix, Hallpike, and Hood [7] reported that speech tests on two cases with eighth nerve neurofibromata showed excellent results at high intensity levels and also found that these same patients could hear speech well through hearing aids in the defective ear. Eby and Williams [8] reported: "Speech intelligibility tests were made in six cases of nerve fiber degeneration. In five of these cases, speech continued to become more and more intelligible with increased intensity of sound, until the limit of the audiometric scale was reached." Schuknecht and Woellner [16] found: "Two human patients with proven acoustic neurinomas were found to have abnormally poor speech discrimination in relation to the pure tone thresholds in the involved ears. In the one case there were normal pure tone thresholds for the three speech frequencies, and a discrimination score of 16 per cent, and in the other case there was an average loss for speech frequencies of 42 db. and a speech discrimination score of zero. Patients having pure-tone threshold losses of similar magnitude from

lesions of the organ of Corti exhibit a less severe loss of speech discrimination." Walsh and Goodman [19] summarize: "The conclusions of this report may be stated on a tentative basis subject to the obvious need for additional data: 1. The relation between threshold loss and discrimination for speech does not differentiate between auditory lesions in the cochlea and those central to the cochlea. At the very least these observations dictate considerable caution in interpreting very poor discrimination for speech associated with moderate threshold loss as being *unrelated* to central auditory lesions."

Obviously, additional research (unbiased and carefully controlled) is needed until greater unanimity of conclusions exists regarding ability to understand speech by patients with hearing impairments resulting from mediocochlear lesions at the different neurologic levels. This brief, incomplete allusion to the diagnostic potentials of the speech tests indicates that the mediotympanic localization implications of such tests are inconclusive. The results of the speech tests at present (and in the future) must be evaluated in relation to the other functional hearing tests, the ear history, and the physical examination.

D. "Mixed" Involvements.—The meaning of the phrase "mixed deafness" was more or less clear and useful in relation to the old dichotomous division of hearing impairments but may be ambiguous in the proposed tripartite classification. Simultaneous involvement of the tympanic and cochlear structures can be called tympanocochlear impairment; of the tympanic and neural structures, tympanoneural involvement; of the cochlear and neural structures, cochleo-neural involvement; of all three levels, tympano-cochleo-neural involvement, and so on. Tympanic pathology plus undetermined mediotympanic involvement could be characterized as tympanomediotympanic until a more exact localization of the mediotympanic pathology is ascertained.

Summary

From the diagnostic viewpoint, the functional hearing tests are employed primarily to ascertain the anatomic localization of pathology which affects the hearing process. A classification of ear or hearing involvements based solely on the anatomic localization of pathology is suggested. The organization of the terminology framework consists of three main categories: (1) tympanic, (2) cochlear, and (3) neural. (The term mediotympanic is useful until accurate localization of the lesion can be confirmed within cochlear or neural tissue.)

This nomenclature is presented for the following reasons: (*a*) this classification scheme is based on a unified concept—anatomic localization of pathology responsible for hearing involvements; (*b*) already familiar terminology is employed in a systematic logical manner; (*c*) the suggested classification is sufficiently flexible to include additional anatomic categories as more refined localization testing techniques become available, and (*d*) it is anticipated that the use of this classification will encourage a more critical and exact determination of the localization of pathology which interferes with the hearing process.

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Chronic Otitis Media and Mastoiditis Due to Proteus Vulgaris (Bacillus Proteus)

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The management of temporal bone infections due to Proteus vulgaris (Bacillus proteus) continues to be a perplexing problem in otology. The success of antiobiotic therapy in treating most infections of the ear is often not evident when this organism is involved. In fact our concern with P. vulgaris has not materially changed since the report [1] made in 1936. This situation is illustrated by four cases in which the usually ubiquitous saprophytic Proteus, flourishing in the confined space of a chronic otitis media extended centrally to produce a fatal intracranial complication.

These cases warrant discussion for a number of reasons. Chronic otitis media is one of the major problems in otology. When P. vulgaris is implicated, because of its insidious nature, the disease becomes more complex. Also there appears to be an increased incidence of Proteus found in routine cultures of all sorts in recent years, perhaps because bacteriologists are paying more attention to the importance of P. vulgaris as a pathogen. Further, considerable interest has been taken in the resistance of strains of Proteus to antibiotics, the development of cross resistance, the change in sensitivity of Proteus to antibacterial agents over a course of years, and the possible effectiveness of combined antibiotic therapy against this organism. Finally, we must consider the problem of the emergence of Proteus as a resistant pathogenic organism in a mixed infection after the sensitive flora have been eliminated by routine antibiotic therapy.

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In 1944 McKee [2] reviewed the literature on P. vulgaris infections and reported a case of septicemia of otitic origin treated with sulfapyridine with recovery. He noted that Proteus is mostly seen in chronic otitis media, but pointed out the difference of opinion as to whether it is the primary causative organism or a secondary invader. He found that only 7 out of 21 reported cases of P. vulgaris otitis media and mastoiditis recovered. He recorded 18 cases of P. vulgaris meningitis caused by otitis media; four of the 18 cases recovered. In the case of brain abscess due to P. vulgaris, four cases were found; two recovered and two died. There were 23 reported cases of septicemia, mostly otitic, due to P. vulgaris; 5 of the 23 recovered.

Birrell [3] stated in 1945 that only 13 cases of lateral sinus thrombosis due to P. vulgaris have been reported, although this type of infection was increasing in frequency. Six of these patients survived the infection. He recorded a case of his own successfully treated with penicillin, sulfathiazole, and surgery.

In 1950 Taylor [4] reported three cases of P. vulgaris mastoiditis and lateral sinus thrombosis with recovery. He noted that seven further cases had been reported in the literature since McKee's article. Three of these seven recovered. Taylor's cases were treated surgically and with streptomycin.

Silcox [5] in 1955 reported three cases of otitis media with serious complications that were not controlled by antibiotics because of resistant bacteria. One case died of P. vulgaris infratentorial abscess and presented the typical picture of a necrotic mastoid, an

antral cholesteatoma which had penetrated the tegmen, perisinus abscess, and lateral sinus thrombosis. Silcox concluded that bacterial resistance to antibiotics is on the increase, and complications of ear infections are being seen more frequently. He urged sensitivity cultures to determine the most effective antibiotic, and complete surgical removal of infected tissue and abscess drainage.

McBean [6] found 15 cases of otitic meningitis treated at the Mayo Clinic between 1946 and 1955. The mortality rate was 40%, but none of the deaths occurred in cases of meningitis with chronic otitis media. Proteus was cultured from three of the chronic cases. He advised surgical management of the otitic focus and antibiotic therapy and chemotherapy.

Freedman, Love, and Hallberg [7] reported a successfully treated case of otitic Proteus meningitis. They point out the rarity of Proteus mastoiditis and meningitis and state that approximately two-thirds of the cases of Proteus meningitis reported in the literature were secondary to otitis media. In their case the typical findings were present—chronic otitis media, cholesteatoma pressing on the facial nerve, dura over the sigmoid sinus covered with granulations, epidural abscess, and meningitis. The organism was sensitive to dihydrostreptomycin, chlortetracycline (Aureomycin), and chloramphenicol (Chloromycetin). The institution of continuous subarachnoid lumbar drainage appeared to be an important factor in the recovery of the patient. The authors felt that the previous treatment of the otitis media by antibiotics eradicated all sensitive organisms, leaving the resistant Proteus.

The four fatal cases of Proteus brain abscess here reported reflect the experience of others as reported in the literature. Otogenic cerebellar abscess is rare and often fatal. Medwick, Uihlein, and Hallberg [8] found only six cases at the Mayo Clinic over a 10-year period. McKee [2] found only four reports in the literature of cases of brain abscess due to P. vulgaris, and a mortality of 50%. Chambers and Clark [9] reported a case of Proteus abscess of the cerebellum of otogenic origin. Their successfully treated case presented the typical Proteus picture: chronic suppurative otitis media treated previously with penicillin, secondary mastoiditis, thrombosis of the lateral sinus, subdural abscess in the posterior fossa, meningitis, and brain abscess. The acute onset of the intracranial complication first necessitated drainage of the cerebellar abscess; a radical mastoidectomy was done later. The patient was given streptomycin, penicillin, chlortetracycline, chloramphenicol, and sulfadiazene at one time or another during the three-month illness.

Morse [10] reported three cases of chronic mastoiditis with intracranial complications—meningitis with lateral sinus thrombosis, temporal-bone abscess, and a fatal case of multiple brain abscesses. A coliform bacillus was identified in the first case, Gram-positive cocci and Gram-negative rods in the second, and a Proteus organism in the third case.

A study of the literature reveals a number of drugs effective in P. vulgaris infection. Waisbren [11] successfully treated a case of severe bacteremia due to Proteus with large doses of penicillin. He regards the treatment of infections due to the Proteus organisms one of the current problems of antibiotic therapy, chiefly because of their resistance to many antibacterial agents, and the problem of Proteus survivorship in diseases where prophylactic chemotherapy is used. He noted that streptomycin, neomycin, chloramphenicol, oxytetracycline, and sulfisoxazole have been reported as effective against Proteus organisms. Some strains respond to penicillin and to a combination of penicillin and chloramphenicol. Waisbren felt that penicillin alone can be used if a rapid bacterial effect is needed or combined with chloramphenicol if a sustained bacteriostatic action is wanted. The statement by Cohn [12] sums up the present concept of Proteus therapy. "Proteus infections are not regularily responsive to any satisfactory treatment regimen. The various species of this genus have somewhat different susceptibilities. Thus about fifty per cent of strains

of Proteus mirabilis are inhibited by moderately high concentrations of penicillin or chloramphenicol. The other species, Proteus vulgaris, Proteus rettgeri and Proteus morganii, are moderately susceptible to the tetracyclines. A variety of other agents, such as mandelic acid, the nitrofurantoins and neomycin, have been reported to have significant degrees of activity against species of Proteus. On the whole, clinically, no agent has been strikingly effective. It would seem worth while to indentify species of Proteus carefully and to be guided in therapy by antibiotic senstivity tests."

Spittel [13] and colleagues noted the resistance of Proteus organisms to the commonly used antibiotics. They found the interesting paradox in seven cases of Proteus bacteremia in which the in vitro studies showed a relative resistance to oxytetracycline (Terramycin) and dihydrostreptomycin, yet combined therapy with these agents was successful in all cases. The expected mortality rate in this type of infection is in excess of 60%. They suggest that the bacteriostasis resulted partly from the patient's serum and partly from the antibiotics used.

Studies in our laboratory have shown that novobiocin, among the newer antibiotics, is moderately effective against many strains of Proteus, and this has been the experience of others.[14]

The question of the effectiveness of combinations of antibiotics has interested a number of investigators, on the theory that the simultaneous use of combinations of drugs would result in a more effective bactericidal tissue dose, whereas the concentration of either drug alone would be ineffective. Armstrong and Larner [15] considered dihydrostreptomycin the best single drug against P. vulgaris, but the results of suboptimal doses of dihydrostreptomycin were improved by the supplement of chlortetracycline or oxytetracycline. Chloramphenicol was found to be of no value as a supplement. No cross resistance or increase in toxic reactions was noted. Pomerantz [16] states that Proteus infections are still highly resistant to drugs, which suggested the possibility of treatment with various combinations of antibiotics. He mentions, however, the fact that drug antagonism has been reported with chloramphenicol-penicillin, whereas Waisbren and Carr [17] found that penicillin and chloramphenicol showed an additive as well as a synergistic effect when the two antibiotics were combined. Goldstein and Daly [18] advised the use of two different antibiotics in order to attack enzyme systems of bacteria in different ways and prevent the development of resistant strains. In the chronic ear, nose, and throat infections they found P. vulgaris the next most commonly encountered pathogen to hemolytic Staphylococcus aureus. They rely on individual culture and sensitivity tests. The combination of sulfisoxazole and oleandomycin in one tablet is said to be effective against Gram-positive and many Gram-negative organisms, including P. vulgaris, with no evidence of cross resistance with most of the antibiotics.

It has been expressed that the question of whether this type of infection is a primary or a secondary one is not important; the pathology caused by Proteus is the significant factor. Experimentally Friedmann [19] injected intra-aurally various organisms grown from recent cases of otitis media. Pseudomonas aeruginosa (Bacillus pyocyaneus) and P. vulgaris consistantly caused acute suppurative otitis media followed by chronic otitis, new bone and cholesteatoma formation. Pneumococcal infections were more virulent, often causing early death due to suppurative meningitis.

Previously Friedmann [20] had studied the incidence of some Gram-negative bacilli and their antibiotic pattern. He cultured Proteus in 87 of 318 cases of chronic otitis media and in 25 of 183 mastoid cases. In addition a variety of Gram-negative coliform bacilli were grown in a number of cases of acute otitis media. In these studies Proteus was usually sensitive to streptomycin and chloramphenicol; only about 20% were sensitive to chlortetracycline.

Regarding the point of view of Proteus as a secondary invader, Yow[21] has described cases of Proteus and Pseudomonas infections that developed during or following antibiotic therapy. He points out the increase in incidence of Proteus and Pseudomonas in routine cultures. He feels that the role of these organisms is similar to that of Candida, and the elimination of sensitive bacteria by antibiotic agents permits the overgrowth of organisms normally present but resistant to therapy. The management of these cases is based on adequate surgical drainage and the administration of drugs found by sensitivity tests to be effective against the particular strain involved. Yow found polymyxin and combinations of streptomycin and large doses of sulfonamides to be the best treatment.

Report of Cases

CASE 1.—A man aged 31 gave a history of having draining ears for many years. Four months before admission to the Memorial Hospital the left ear began to pain and discharge profusely. A radical mastoidectomy was performed in another city. The postoperative course was complicated by meningitis, which responded to penicillin therapy, and left facial paralysis. Because the retroauricular pain persisted, he decided to return home for further treatment. Examination showed a left peripheral facial palsy, the dry radical mastoidectomy cavity on the same side, and a chronic suppurative otitis media on the right side. The contents of the middle ear on this side were largely destroyed, and the cavity was filled with necrotic debris and cholesteatoma. The neurological examination and the physical and laboratory examinations were not contributory. The left facial nerve showed a partial reaction of degeneration to faradic stimulation.

Because of the pain and the facial palsy, exploration of the mastoid was advised. The operative findings were negative; the facial canal was decompressed. The postoperative course was uneventful except for moderate elevation in temperature and considerable pain, vertigo, and nausea. Under chloramphenicol therapy the temperature subsided and his symptoms largely disappeared.

Following his discharge from the hospital he continued to improve. The pain disappeared, but he still had the facial palsy and some vertigo and nystagmus, which were considered labyrinthine in origin. Seven weeks after his discharge he complained bitterly of increasing occipital pain. Hospitalization was advised, but he did not return to the hospital until the following day.

On admission the patient was obviously in great pain. He was given 100 mg. of meperidine (Demerol) and 25 mg. of chlorpromazine (Thorazine), and fell asleep in a short time. An hour or so later the nurse noted that he was not breathing, and closer examination showed he had died.

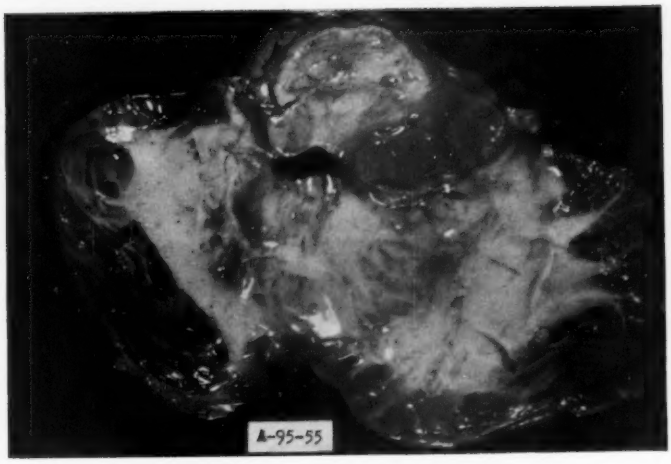
Postmortem examination showed a meningitis and left cerebellar abscess due to P. vulgaris (Figure). He also had a large foul-smelling cholesteatoma in the right mastoid bone.

CASE 2.—A 36-year-old man was admitted to the University of Virginia Hospital on Jan. 6, 1950, as an emergency. He had been hospitalized in a nearby hospital for a draining left ear. In spite of penicillin treatment the process failed to improve, and he was referred as having developed a brain abscess. On admission he presented the appearance of an acutely ill patient, dehydrated, and incoherent. The left ear showed a destroyed drum, a foul-smelling discharge, and a middle ear filled with granulations. His pupils were equal and reacted slightly to light; all tendon reflexes were hyperactive. The outline of the left optic disk was blurred. The examination of the urine was essentially negative. The spinal fluid showed total proteins 210 mg., chlorides 715 mg., and sugar 60 mg. per 100 cc. The white cell count was 18,120, with 87% polymorphonuclears. The culture of the ear discharge was reported later as showing P. vulgaris. The x-ray of the mastoid showed invasion of the tegmen tympani and possibly an overlying brain abscess. Medical and neurosurgical consultations concurred in the opinion of an otogenic brain abscess. Active penicillin and streptomycin therapy was instituted.

A radical mastoidectomy was performed on the day of admission. The roof of the middle ear was found diseased; it was removed, and a tract leading to a large temporal lobe abscess was aspirated. Fifty cubic centimeters of a thick foul-smelling pus was withdrawn. A drain was left in place. A few hours after surgery the patient died.

CASE 3.—A 13-year-old girl was admitted into the University of Virginia Hospital on Sept. 14, 1952, as an emergency. She gave a history of a draining ear for the past year. Her local physician had treated the ear with good results until two months prior to admission, when she developed a foul discharge from the left ear, spiking temperatures, and episodes of pain. Ten days before admission she had a simple mastoidectomy. The lateral sinus was found involved, and a culture of P. vulgaris was grown from the free pus encountered in the sinus. The patient did poorly postoperatively in spite of the use of antibiotics.

On admission to the University of Virginia Hospital the patient appeared quite sick; her tem-



Cerebellar abscess found at autopsy. (Case 1)

perature was 103 F and pulse 130. Because of nuchal rigidity and a dilated left pupil a spinal tap was done which showed 500 cells; no organisms were found. On the second day after admission a radical mastoidectomy was performed. The mastoid antrum was found full of pus; the dura of the middle and posterior fossa was exposed and found covered with granulation tissue. She was given 500,000 units of penicillin every two hours, 0.5 gm. of streptomycin and 750 mg. of chloramphenicol every eight hours. Sulfadiazine was given by intravenous drip. The postoperative course was progressively downhill. The meningitis increased in severity; daily spinal taps showed an increasing purulent change in the fluid. Culture of the spinal fluid showed P. vulgaris, sensitive only to chloramphenicol. She developed a potassium deficiency and died in coma two weeks after admission.

CASE 4.—A 27-year-old man was admitted to the Memorial Hospital as an emergency. Two weeks before admission he developed severe headaches and vomiting and a flare-up of his left ear, which had drained on and off for many years. On admission the chronic middle ear infection was noted, and marked nuchal rigidity was demonstrated. Spinal fluid examination showed 1610 morphonuclears, 380 lymphocytes; sugar 75 mg., protein 104 mg. per 100 cc. An endaural radical mastoidectomy was performed. The middle ear was filled with pus and granulation tissue. A large pathological exposure of the sinus plate, perisinus abscess, and a thrombosis of the lateral sinus were found. The culture of the pus from the mastoid was reported as P. vulgaris. The postoperative course was stormy. The patient was given chloramphenicol, streptomycin, and sulfadiazine in adequate doses. The patient complained of considerable pain, and the temperature remained elevated. There were no focal signs of brain abscess. Because of his unsatisfactory progress and the development of generalized convulsions, he was transferred to the University of Virginia Hospital.

The salient finding on admission was a right homonymous hemianopsia; other than a draining, deaf left ear, the physical, neurological, and blood and urine examinations were essentially normal. The spinal fluid was slightly turbid and contained 142 cells with 96% lymphocytes. X-ray of the mastoid and petrous bones was nondiagnostic except for the surgical defect. The electroencephalogram showed an abnormal record with a focal emphasis on the left mid and anterior temporal fossa. The patient was continued on streptomycin, chloramphenicol, and sulfadiazine therapy. Two days after admission a revision of the radical mastoidectomy was done. The attempts to find a cerebral abscess through accessory burr holes were unsuccessful.

The postoperative course was uneventful; he improved rapidly. Ten days after operation the nurse heard the patient take a deep sigh. When she came to see him, he had died.

Postmortem examination showed an abscess between the calcarine fissure and the falx cerebri, suppurative meningitis, and thrombosis of the left lateral sinus. Culture showed P. vulgaris.

To allay the impression of fatality in this type of infection a successfully treated case of P. vulgaris chronic otitis media and mastoiditis with a complicating meningitis is reported.

The patient was a 15-year-old boy who had a simple mastoidectomy performed five years before the onset of his present difficulties. Three weeks before he was first seen, the ear that had been operated on flared up, and he developed a headache and stiff neck. Examination showed a foul-smelling otorrhea, from which a culture of P. vulgaris was grown. The cerebrospinal fluid showed 300 cells. He was treated with penicillin and novobiocin. The headache, stiff neck, and ear discharge disappeared. He was referred to the University of Virginia Hospital for surgery. At operation a large cholesteatoma was found filling the mastoid cavity. The cholesteatoma had eroded the sigmoid plate, but the sinus was not thrombosed. Culture from the ear showed P. vulgaris sensitive to chloramphenicol. The administration of penicillin and novobiocin was continued, with the later addition of chloramphenicol. Except for a temporary increase in the spinal fluid cell count, recovery was uneventful, and the patient was discharged 10 days after operation.

Comment

From a clinical point of view P. vulgaris otitis media and mastoiditis is usually characterized by extensive destruction of the middle ear and mastoid, fetid otorrhea, cholesteatoma formation, granulation and necrotic tissue, pathological exposure of the lateral sinus and dura, and epidural abscess. Otitic facial palsy is not infrequent.

The onset of intracranial extension—lateral sinus thrombosis, septicemia, meningitis, and brain abscess—is often rapid. Otalgia, headache, stiff neck, vomiting, fever, and convulsions initiate the symptomatology. Early spinal puncture is essential.

The medical and surgical management of cases of chronic otitis media in which P. vulgaris has been identified as the causative organism is the same as the accepted treat-

ment of chronic otitis media caused by any other bacteria, with the exception of an added amount of alertness to the treacherous propensities of Proteus. We are inclined to regard the main treatment of Proteus infections of the ear, with or without complications, as surgical. The newer concepts of reconstructive surgery of the middle ear will promote earlier and more complete operations for this type of chronic otitis media. In this way the serious complications will be avoided; the functional capacity of the ear will be maintained, and a dry cavity assured. We have followed the clinical classification of Lillie in the management of chronic middle ear suppuration. X-ray studies are of paramount importance. Cases of chronic otitis media of the nondangerous type, with a central perforation, are treated conservatively; after the culture and sensitivity tests have been obtained, the drug of choice is used locally and systematically. Surgery is advised if a cholesteatoma can be demonstrated; if pain, vertigo, or nausea is a factor; if a fistula sign is evident, or if the progress under conservative management is not promptly satisfactory. The surgery should be meticulous and complete. The postoperative period should be closely observed, and prompt attention should be paid to symptoms and signs of intracranial involvement. There should be close cooperation between the otologist and the neurosurgeon.

The antibiotic therapy should be in adequate dosage and prolonged, and with the realization that antibiotics are ineffective in walled-off necrotic temporal bone infections and encapsulated brain abscesses. The present trend is toward combinations of antibiotics. In the acute case, before the sensitivity tests are reported, chloramphenicol should be the drug of choice. This agent is often effective against strains of Proteus and possesses a high diffusion and penetration through the blood-brain barrier.

In reviewing his cases of otorhinogenic intracranial infection, Harpman [22] points out the experience of most otologists—difficulty in accurate diagnosis because of signs and symptoms masked by antibiotics. He points out that successful management requires assessment of each case individually. For example, often in the presence of suppurative lateral sinus thrombophlebitis, the symptoms and signs of fever, chills, metastatic suppurative foci and elevated white count may be absent. He points out the futility of treating chronic otitis media, especially with cholesteatoma, with antibiotics and sulfonamides. Otitic meningitis may not always be secondary to chronic middle ear infection but may complicate an otitis brain abscess, and treatment of the abscess is the main factor in recovery. The history is a main point in the diagnosis—headaches, periods of aphasia, vomiting and loss of weight, neck stiffness and convulsions in temporal lobe abscess. Timely surgery is necessary if life is to be saved and permanent disabilities avoided. Delay in operation, waiting for encephalation, is no longer valid when antibiotics are available. If a brain abscess is suspected, all aids to diagnosis, including electro-encephalography should be used. Selection of the appropriate antibiotic is paramount.

In the preantibiotic days intracranial infections had an extremely high mortality rate. Since then the prognosis has considerably improved. Pennybacker [23] found in his cases of brain abscess, of all types, the mortality dropped after penicillin from 46% to 29%. From the years 1938 to 1949 he observed 110 cases of brain abscesses, 56 from the paranasal sinus and mastoid. Of these, 10 cases originated from the sinus, with a mortality rate of 20%, and 46 cases from the mastoid, with a mortality rate of 37%. Ballantine and White [24] surveyed the brain abscess cases seen in their hospital. In the five-year period, 1936-1940, 10 cases of otogenic brain abscess were seen, and the mortality was 67%. In the period 1946-1950 17 cases were seen, and the mortality was 30%. The increase in incidence of brain abscess in the antibiotic era is attributed to

the reduction in immediate mortality with antibacterial therapy. More patients survive the acute illness and proceed to intracerebral suppuration.

On the other hand, Gurdjian and Webster[25] found ear infection not as important as formerly in their series of 45 cases seen in the last 12 years. No cases of P. vulgaris infection were recorded in their article.

Conclusion

The treatment of temporal-bone infection in which Proteus vulgaris is the offending organism still constitutes a challenging problem in otology. The successful management of this type of infection is based on its early recognition, persistent antibiotic treatment, close observation for mastoid and intracranial complications, and prompt mastoid, middle ear, and brain surgery, when indicated. The concept of tympanic plastic operations will contribute largely to the solution of this problem.

Forest Hills (Dr. McGovern).

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Head and Neck Pain in Temporomandibular Joint Disease and Muscle Spasm

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History

Pathological changes in the temporomandibular joint of early modern man have been reported.[1] The surgeon of the Old Kingdom of Egypt, 3000 B. C., knew the anatomy of this joint. The earliest known surgical text, the Edwin Smith Papyrus,[2] has one case, a "dislocation of the mandible," with the method of reduction that is still used today. In 1842, Cooper is said[3] to have reported snapping in the temporomandibular joint, and in 1887 Annandale[3] repositioned the articular disc and sutured it to the outer side of the joint. Lang has been named[3] as the surgeon who introduced disc extirpation (1909). The role of the dental apparatus in temporomandibular joint disturbances was first noted by Prentiss[4] and Summa[5] in 1918. Costen[6-10] and Goodfriend[11-13] in the early 1930's called attention to the diversity of symptoms produced by pathology of the temporomandibular joint, and Costen[9,10] in 1939 first noted the importance of spasms of the mandibular musculature.

Anatomy

There is still much to be learned about the anatomy and physiology of the the temporomandibular joint and the mandibular musculature. Sicher[14-17] and Shapiro[19,20] have contributed a great deal to our knowledge. The basic anatomy of the joint is well known, and its description is best left to the standard textbooks of anatomy. Certain facts, however, deserve special notice because of their implications.

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The temporomandibular joint is a ginglymodiarthrodial joint formed by the articulation of the condyle of the mandible with the articular disc, the articular fossa, and the articular tubercle. There are great variations in the size, shape, degree of wear, and position of the condylar heads.[19,21] There is even great variation between the two condyles in the same person.

The articular disc is specialized connective tissue, not fibrocartilage as is often stated, and this fact would indicate that it is capable of repair. Disc perforations occur very rarely. In studies[19,22] of 1100 discs of human cadavers, only one perforation was found. The disc is fused medially, anteriorly, and laterally to the articular capsule. Posteriorly, it is connected to the capsule by loose, highly vascular connective tissue and is supplied with nerves from branches of the auriculotemporal nerve. The disc and its attachments serve to divide the joint into upper and lower joint cavities, which are lined by synovial membranes richly supplied with nerves, blood vessels, and lymphatics.

The capsular ligament is a thin, loose fibrous sac with the temporomandibular ligament on its lateral side. The stylomandibular and sphenomandibular ligaments are actually fascial structures. The ligaments merely limit the movements of the elements of the joint. It is the muscles that maintain the joint elements in their position.

The auriculotemporal and chorda tympani nerves are in such positions that they cannot be affected by a displaced condyle, nor can a posterior displacement of the condyle compress the Eustachian tube or interfere with the action of the tensor palati muscle. Only if the postglenoid proc-

ess were destroyed, could the condyle impact and erode the tympanic plate. Shapiro [19] and Sicher [17] have failed to find any significant erosions of the tympanic plate in the cadavers they have examined. Sicher [23] concludes: "Even if all defects in the lateral part of the tympanic plate are considered as caused by articular changes, this condition is extremely rare, is restricted to the oldest age group and cannot in any way be related to disturbances of hearing."

Joint Action

The actions of the temporomandibular joint are most complicated; so much so, in fact, that concentrated research for the past 20 years has only begun to reveal them in all their complexity. It should always be remembered that mastication represents only a very small part of the daily work the joint performs. For example, in swallowing, which is estimated to occur every 60 to 90 seconds, the mandible rises, moves posteriorly, and then drops to its postural position. There are many physiologic actions which actively involve the joint, such as speaking, coughing, moistening the lips, and yawning; and there are also the pathologic actions, such as clenching the jaws and grinding the teeth.

Basically, the temporomandibular joint has two motions:

1. Ginglymoid, or hinge: This takes place in the inferior synovial cavity, the smaller of the two, and represents the relationship between the condyle and the articular disc.

2. Arthrodial, or gliding: This takes place in the superior synovial cavity. The articular disc and condyle move as a unit down the posterior surface of the articular tubercle and out onto its eminence.

Roentgen motion pictures of the joint are now possible through the use of image intensification.[24] They open a new means of study which should lead to a knowledge of joint action such as we have never had.

Mandibular Musculature

In temporomandibular joint actions, the neuromuscular apparatus governs all movement. The muscle activity is regulated by neural reflexes which originate in proprioceptive nerve endings in the muscles themselves, the articular capsule, and the periodontal ligaments of the teeth. It should be emphasized that in functional movements no one of the muscles acts alone. The mandibular muscles function physiologically as a unit in a smooth, well-related, and split-second action. In a pathologic situation, of course, these statements no longer hold true.

The mandibular musculature can be divided into two groups: (1) the muscles of mastication: masseter, temporal, internal pterygoid, and external pterygoid; (2) the suprahyoid muscles: digastric, stylohyoid, mylohyoid, and geniohyoid. Our concern is chiefly with the muscles of mastication, four powerful muscles which are supplied by the motor branches of the third division of the trigeminal nerve.

The masseter muscle forms a rectangle originating on the zygomatic arch and inserting on the lateral surface of the ramus of the mandible. The muscle fibers are relatively short and lie at an angle to the long axis of the muscle, thus producing a muscle of great power. It is a powerful elevator of the mandible which can also produce some retrusion.

The temporal muscle has a wide origin in the temporal fossa on the lateral surface of the skull. It converges on the opening between the zygomatic arch and the lateral surface of the skull, the insertion being the coronoid process and anterior border of the ramus of the mandible. The muscle fibers are long and follow the long axis of the muscle, thus producing a muscle of movement rather than of great power. It is an elevator of the mandible, but it can also produce retrusion.

The internal pterygoid muscle is akin to the masseter muscle anatomically and physiologically. It is a rectangular muscle which originates in the pterygoid fossa and inserts into the medial surface of the angle of the mandible. The muscle fibers are relatively

short and lie at an angle to the long axis of the muscle, thus producing a muscle of great power although somewhat weaker than the masseter muscle. It also is an elevator of the mandible.

The external pterygoid muscle has two heads. The inferior head, the larger of the two, arises from the lateral surface of the lateral pterygoid plate. The superior head arises from the greater sphenoid wing. The two heads fuse posteriorly and insert into the anterior surface of the articular capsule of the temporomandibular joint and the anterior surface of the neck of the mandible. The muscle fibers are long and follow the long axis of the muscle, thus producing a muscle of movement. The two external pterygoid muscles acting together pull the mandible and the articular discs forward. Acting individually, the external pterygoid muscle will pull the attached condyle forward, downward, and inward.

Muscle is capable of acting in two ways: 1. In isotonic contraction, the muscle tenses and shortens, thus acting as a mover. 2. In isometric contraction, the muscle tenses but maintains its length, thus acting as a stabilizer.

Degenerative Temporomandibular Joint Disease

Degenerative temporomandibular joint disease is similar to degenerative disease in any other joint. However, the articulating surfaces of the temporomandibular joint are covered by fibrous tissue and not by hyaline cartilage as in other articulations. Abrams,[25] speaking medically, states that degenerative joint disease is present universally after 40 years of age, although only 5% present clinical symptoms. Various investigators[26-28] have reported that 28% to 40% of persons show symptoms of temporomandibular joint disturbances, but the absence of uniform diagnostic standards and definitions make impossible any scientific evaluation of these statistics.

Degenerative joint disease is related to the physiologic process of aging, and the repeated trauma, stresses, and strains that occur with time and use are the chief causes. Heredity also appears to play a role, and possibly endocrine disturbances. In another article[29] I list as follows the local factors which produce this condition in the temporomandibular joint:

1. Microtrauma with prematurities and other forms of traumatic occlusion.

2. Overclosure of the mandible in which the condyle suffers a posterior displacement, impinging on the loose connective tissue which connects the posterior border of the articular disc to the articular capsule, eventually causing a degeneration of this tissue and a breakdown of the entire articulation.

3. Hypertension of the mandibular musculature, trismus, and bruxism. Muscular spasms can throw enough pressure of themselves on the joint to do damage. Once the muscles are in a state of contractions, there is a strong tendency to pull the articular disc forward, and the condyle backward, to produce damage by impinging on the loose connective tissue attaching the posterior border of the articular disc to the articular capsule. Muscular spasms may be produced by emotional factors, and both traumatic occlusion and overclosure of the mandible seem sooner or later to be capable of throwing the mandibular musculature into a spastic state.

4. Age. Degenerative joint disease develops with increasing age, starting in the second decade of life, but it may be symptomless. Bauer[30] examined five patients clinically before death and then did a microscopic examination of the temporomandibular joint post mortem. He found that the joint accommodated to pathologic function so well that even where there was almost a complete destruction of the disc there were no marked local or reflex symptoms.

5. Heredity, a factor whose importance has yet to be determined.

There is no simple etiologic factor, but combinations of the various factors.

The symptomatology in degenerative temporomandibular joint disease can be divided into two sections. Firstly, there are the symptoms of the joint disease itself, and secondly, there are the symptoms of the muscle spasms, which may be one of the causes of the disease or which may result from it.

The primary symptom which arises purely from the joint disease is pain in the joint on motion, often getting worse on prolonged

activity. This is usually an aching type of pain which may be referred to adjacent regions. It is relieved by rest, although after prolonged rest, as in sleeping, stiffness is common. There may be some swelling or tenderness over the joint, and clicking, snapping, or grating sounds may be heard.

Muscle spasms will produce their typical pain which may be referred almost anywhere in the head and neck, including the tongue and throat.

Mandibular Muscle Spasms

Mandibular muscle spasms are a serious problem occurring in a syndrome having three outstanding symptoms: (1) pain, (2) mandibular dysfunction, and (3) emotional disturbance. These symptoms are always present, but in any particular patient they are found in varying degrees.

The pain varies widely and is myofascial in origin. It may build up slowly to a considerable intensity and remain at this level for hours or days, or it may be a dull constant type of pain which is most persistent. It is more often unilateral than bilateral, and often tender areas can be palpated in the muscles. Palpation of these areas may even produce referred pain in the joint.

Mandibular dysfunction is seen constantly, but the degree varies widely. The patient may present with an inability to open his mouth more than a few millimeters or the amount of opening without pain may be only 5 or 10 mm. less than normal. In many patients, the mandible deviates to one side as the patient opens his mouth. I have recently observed a patient referred for pain in the zygomatic region who first showed a marked deviation to the right and then on approaching full opening the mandible moved farther to the left than it had originally to the right.

Typically, the patients who present with these mandibular muscle spasms have emotional disturbances to a greater or less extent, and the connection is rather obvious. From earliest infancy, the mandibular musculature is used to express emotions, as in crying, accepting or rejecting food, smiling, and expressing love or hostility through sounds and later speech. We also have the organ language to show this connection, as choking down anger and gritting one's teeth. In fact, I believe that the mandibular muscle spasms in these patients is on an emotional basis. For this reason, the reassurance of the patient is emphasized in treatment and appears to be essential for successful therapy.

Differential Diagnosis

The symptoms of degenerative temporomandibular joint disease and mandibular muscle spasms have been fully described earlier in this article and in other articles by me.[29,31] Two facts complicate the diagnosis: The first is that the pain of this joint disease can reflexly throw the mandibular musculature into spasm; the second is that mandibular muscle spasms will sooner or later produce a degenerative joint disease. The old question of which came first, the chicken or the egg, now assumes vital importance. If the joint disease is the basic disorder, clearing up or even alleviating the pain of this condition will bring the mandibular musculature out of its spastic state and prevent its recurrence. However, if the muscle spasms are the basic disorder and have damaged the joint, the patient must be treated for both conditions which at this stage are united in a vicious self-perpetuating cycle in which each increases the intensity of the other.

The clinical history is of the first importance in making a differential diagnosis. The emotional status of the patient, while an important consideration here as always, must be kept in its proper proportion, for there is no reason to assume that emotional disturbances must manifest themselves in mandibular muscle spasms. Mandibular dysfunction also must be evaluated with care because it can and does occur in both these conditions, and here again a careful history will be of great help.

Roentgenography is of limited diagnostic value for two reasons. Firstly, roentgenographic evidence of arthritis does not neces-

sarily mean that the patient will present symptoms of this condition. On the contrary, we know that the great majority of these conditions are symptomless.

Secondly, anatomic considerations present many distinct problems in accurately and completely visualizing this joint roentgenographically. Arthrography makes it possible to visualize the synovial cavities but tells us nothing else. Laminagraphy gives a roentgenographic visualization of a prescribed plane with a width of cut approximately 0.5 to 0.75 cm. Cinefluorography with image intensification [24] is a new technique that may have a great deal to offer. Probably the best technique is the same cranial-oblique technique that is used for the mastoid bone.

Roentgenograms of the temporomandibular joint show the condyle and the articular fossa in profile. They cannot visualize the general shape of the condyle, its inclination, its relation to the articular disc and fossa, or the size of the joint spaces, and of course no information is obtainable about the articular disc. The same problems present themselves in the study of the articular fossa and the articular eminence. However, the roentgenograms can show evidence of arthritis, but I should hesitate to rule out degenerative joint disease on the basis of a negative roentgenogram.

Treatment

Basically, the treatment of degenerative temporomandibular joint disease is that of any joint with the same condition. The general medical treatment as outlined by Abrams [25] is primarily a matter of rest and avoidance of trauma, strain, and any abnormal usage. He prescribes heat, massage, and analgesics (acetylsalicylic acid and its compounds). In temporomandibular joint disease we must look to the dental apparatus for the source of any trauma. Traumatic occlusion and pathologic loss of vertical dimension must be corrected. With age there is a physiologic loss of vertical dimension, which must not be altered nor should it be confused with pathologic loss. Altering a healthy vertical dimension will only lead to a degeneration of the joint. Moist heat, analgesics, and limitation of use are important forms of therapy. Where the pain has reflexly produced muscle spasms, moist heat and analgesics will give prompt relief, and if the joint disease is relieved the muscle spasms will not recur.

In mandibular muscle spasms, we are faced with a different problem. The deep-seated emotional problems which are usually present can best be treated by psychotherapy, but in most cases this is either unacceptable to the patient or financially impossible. We must therefore fall back on physical therapy and such psychotherapy as we can offer. Getting the patients to talk is most important, for it will give them an opportunity to express their fears and anxieties about this condition and thus open the way for us to give them the reassurance they need. Talking may also give the patient the opportunity to work out or at least indicate some of the other problems which are behind this somatization and make it possible for us to help him. The reassurance of a definite and hopeful diagnosis is of the greatest importance to these patients.

To relax the muscle spasms, several methods are available. I have found ethyl chloride sprays in the office and moist heat at home the methods of choice. The muscles affected are sprayed with ethyl chloride in long, slow, sweeping movements so that the skin is not whitened. This is continued until the pain is relieved. With the disappearance of the pain, the mandibular dysfunction is usually decreased or may disappear entirely. The patient is then instructed in the use of moist heat, to be applied two to four times a day for periods from a half-hour up to an hour. Wool or flannel cloths are soaked in hot water, wrung out, and applied to the areas designated. These treatments do not directly affect the internal or external pterygoid muscles, but treatment of spasms of the masseter and temporal muscles seems to clear up spasms of the other muscles.

The new muscle relaxants are useful where the patient cannot use the moist heat. Tetracaine hydrochloride and procaine hydrochloride [33,34] have been injected into the tender areas in the muscles and have also proved successful.

I have one patient who reported attacks of increasingly severe pain over the "whole side of the face and head" for a period of six to eight months. The pain caused her to seek dental care, but clearing up the dental pathology did not stop the pain. It was possible to examine the patient during the severest episode of pain, which at this time also included pain down the back of the neck and into the shoulder. Examination revealed a spasm of the left masseter muscle with a distinct tender area close to the middle of the anterior border of the muscle, and a limited mouth opening of 30 mm. Spraying the masseter muscle with ethyl chloride gave immediate relief from the face and head pain, and the mouth opening was increased to 40 mm., which is approximately the normal for this patient. The whole matter was explained to the patient, and the relief of the pain was her proof. A muscle relaxant was prescribed and she was instructed to use it until all pain was gone for 24 hours, and then 30 minutes before every dental visit and any time the pain should recur. It has been six months since this visit, and the patient reports that she has not used the drug since the initial dosage and has had no recurrence of the pain. Here two things seem to have cleared up the condition. First is the reassurance given by a full explanation with a proved diagnosis, and second is the understanding of the emotional basis of this condition by the patient.

With the disappearance of the muscle spasms, we may find the patient complaining of joint difficulties. Where the muscle spasms have been present over a period of time, degenerative joint disease can be produced. The muscle spasms limit the movements of the mandible and so mask the symptoms of the joint disease, while the pain of the muscle spasms overshadows any other pain. The joint disease must now be treated as we outlined above.

Prognosis

Only in hip joint involvement does any real deformity or crippling result from degenerative joint disease. In a series of 515 patients, Russell and Bayles [34] did not see a single case of complete ankylosis of the temporomandibular joint. Complete ankylosis occurs rarely if ever in temporomandibular joint arthroses. With proper treatment, the prognosis is favorable and we can look forward to a good functional result.

The prognosis in mandibular muscle spasms is also favorable. Most patients with this problem can obtain relief but they may continue to grind or clench their teeth, thus always making it possible for the muscles to be thrown into spasm. However, the patient has the means of controlling these spasms.

Summary

The temporomandibular joint is a ginglymodiarthrodial joint whose anatomy and actions are still not completely understood.

The mandibular musculature consists of eight muscles which function as a unit regulated and controlled by neural reflexes.

Degenerative temporomandibular joint disease is similar in etiology, pathology, symptomatology, and treatment to the same condition in any other joint.

Mandibular muscle spasms present problems in symptomatology and treatment and can also produce a degenerative joint disease.

The clinical history is of the first importance in making a differential diagnosis. Roentgenography is of limited diagnostic value.

The prognosis in both conditions is favorable.

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The Schwartze Mastoidectomy

Its Value at the Present Time

WILLIAM McKENZIE, M.B., B.Chir., London

It has been the general experience all over the world that antibiotics have sharply reduced the number of cases which need a Schwartze mastoidectomy.

My own experience in 1951 was that 12 cases needed operation, out of 161 patients admitted to hospital. Even this number is probably too high, and includes some unnecessary operations, as I hope to show later, but it forms a strong contrast to the figures published in 1924 from the Edinburgh Royal Infirmary, when 87 operations were performed in 249 cases. I have chosen these two dates because penicillin was generally used in 1948, whereas in 1924 neither penicillin nor sulfonamides had been discovered.

Before going into more detail, I should perhaps describe the conditions under which I work, because there is no doubt that the number of mastoid operations which are needed depends on the general intelligence and standard of living of the population, and the manner in which a discharging ear is regarded. I work for the most part at the Royal National Throat, Nose and Ear Hospital, London. There were 38,000 new patients at this hospital in 1955, and 190,000 attendances, so the work is considerable and deals with the average population of London as a whole, and at a time when the National Health Service is fully working.

The graph (Fig. 1) shows the number of Schwartze mastoidectomies over the last six years. It will be seen that the fall is continuous and that it has not indeed apparently reached its minimum. This paper

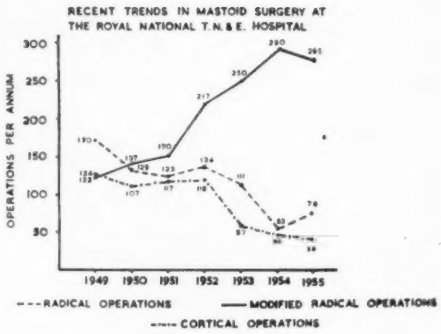

Figure 1

simply speculates on what may happen to this graph in the next 10 or 15 years.

There are three main factors which lead to a recommendation of a Schwartze mastoidectomy; first, of course, is the efficacy of the operation in acute mastoiditis, and I suppose we have all performed a Schwartze mastoidectomy and have been surprised by the rapid recovery of the patient afterward, for nowadays the ear is often dry and the postaural wound completely healed in a week or 10 days. The only doubt remaining is whether the operation was necessary at all. I might perhaps say that with the case notes which I have examined, and which are usually my own, this doubt has often arisen in my mind. These operations are usually undertaken at my hospital by registrars, who are assistants in training, and their experience and skill cannot be considered equal to that of a fully trained surgeon; yet the general result of operation is excellent, with a dry ear and healed drum (Fig. 6). The older operation of revision of a Schwartze mastoidectomy, so familiar to those of us who were trained before the

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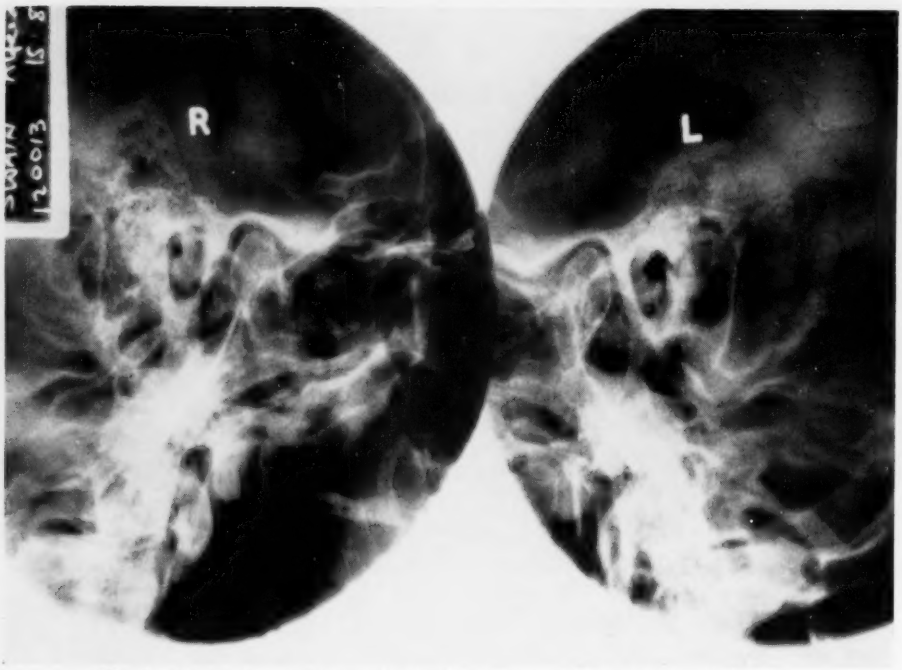

Fig. 2.—X-ray of mastoid process showing cells remaining after mastoidectomy.

use of penicillin, is nearly unknown and yet (Figs. 2 and 3) these few cases, which I have chosen because they have good x-ray photographs, clearly show that some mastoid cells have been left behind by the surgeon. This leads to the problem of what is actually done at operation. The bone chips from the mastoid are often sterile when an attempt is made to produce a bacterial culture from them, so the analogy of removal of a focus of disease or of drainage of an abscess is quite false. Perhaps the removal of tissue with a poor blood supply leads to rapid recovery, even if a small number of diseased cells are left behind.

The efficacy of the operation is, however, not everything; otherwise our curve would not have fallen so steeply. It would be fair, therefore, to glance at the objections to operation, both on the part of the surgeon and on that of the patient. The surgical objections to Schwartze mastoidectomy are real, and familiar to the older surgeons. They are damage to the soft tissues of the meatus and stenosis with the annoying complication of recurrent otitis externa, injury to the ossicles, especially dislocation of the incus with subsequent deafness, and a persistent sinus in the postaural wound. This list does not include the disaster of damage to the facial nerve, which has always been rare in our experience, nor does it include the complications resulting from anesthesia, and later complications from damage to the muscles attached to the mastoid process. This last has often been the cause of complaint in patients, and the pain cannot be distinguished from an ordinary fibrositis.

Apart from all these, there is the simple fact that a patient prefers to avoid an operation if he can do so, and this in the days of antibiotics has undoubtedly led to the sharp fall in the number of mastoidectomies.

The next factor is the mind of the doctor. I put it in this way, because I can often see the mind of the doctor when reading through the case notes. In the first place, most of the cases which need operation

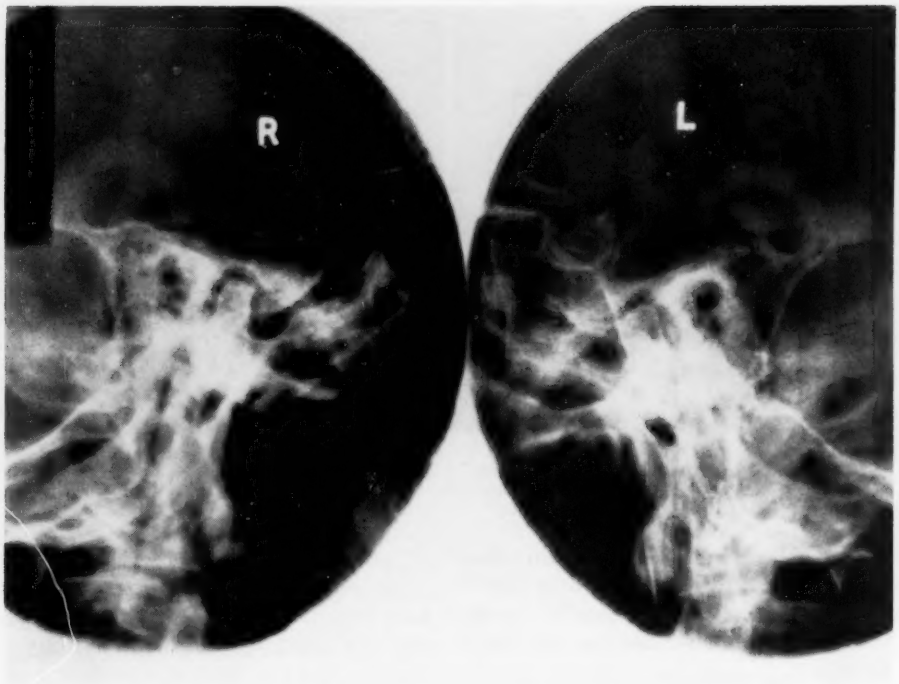

Fig. 3.—X-ray of mastoid process showing cells remaining after mastoidectomy.

have not had penicillin or any other antibiotic until they reach us. This may have been the fault of the patient, who did not ask for advice when the ear began to discharge, or it may have been due to the general practitioner, who is often reluctant to use a needle and syringe, especially when the patient is a child. At all events, the problem which presents itself to us is an untreated otitis media, and the choice lies between antibiotics and mastoidectomy. The commonest fault, in fact, is that antibiotics have not been given at all, or, if they have, they have not been used for long enough (Fig. 5). It is interesting that the fault is seldom that penicillin has been wrongly ordered for an infection by a penicillin-resistant organism. This neglect of antibiotics has, in truth, not been confined to the general practitioner, for sometimes an acute otitis media has been mistaken in our clinics for an otitis externa at the first attendance and no antibiotic has been given until the diagnosis has been corrected. The next factor, the mind of the surgeon, concerns me more closely. Most of us have learned our work before penicillin was used, and we are reluctant to abandon the rules laid down in those days as indications for mastoidectomy. I read through the case notes with these indications in mind; I can sympathize with the surgeon's decision to operate, even if I do not agree with it.

To express this better, I have summarized the indications for mastoidectomy from the most recent textbook I could find. They are from Ballenger's textbook of Otology, fourth edition, 1954.

Indications for Operation

1. A Subperiosteal Abscess.—Figure 4, from Moffett's paper, shows that a subperiosteal abscess will readily subside when penicillin is used without any other treatment. As we all know, incision of the abscess alone with penicillin, or a mastoid-

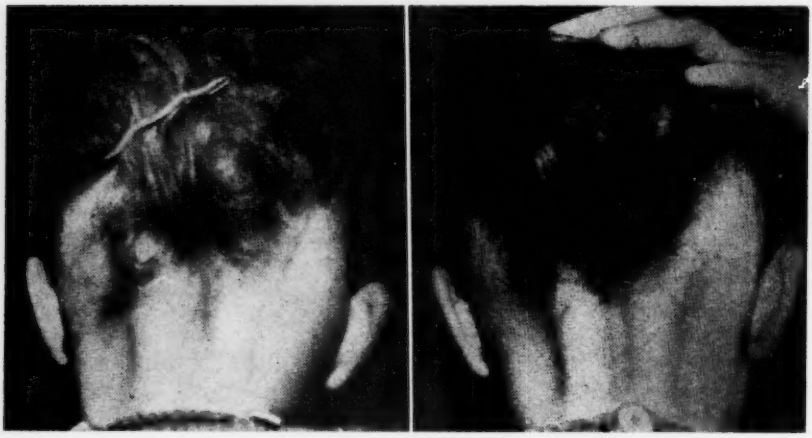

Fig. 4.—Showing the disappearance of a subperiosteal abscess after penicillin treatment. (Reproduced with permission from Moffett, A. J., and Dalton, G. A.: Acute Mastoiditis in Children Treated with Penicillin and Sulphadiazine, Brit. M. J. 2:1087, 1949, published by the British Medical Association, London.)

ectomy and penicillin, or penicillin alone will produce a cure, and it is only fair to say that a subperiosteal abscess, to say the least, is not a certain indication for operation.

II. Persistent Otorrhea for Six Weeks after Adequate Treatment.—The difficult word here is "adequate." How long should antibiotics be used before the decision to operate? We are often faced with this difficulty. A patient who, for example, has had an otorrhea for six months with an acute onset, presents himself at the outpatient service, and the organism is found to be sensitive to penicillin. The mastoid x-ray shows a cellular mastoid. We have been in the custom of using antibiotics daily for a fortnight at least, and many of these unpromising cases have recovered and the ear has remained dry. This leads to speculation about treatment when the mastoid process is sclerotic in the same situation. Experience before antibiotics has shown that an acute mastoiditis may occur in a sclerotic mastoid process, and the infection may disappear after Schwartze mastoidectomy, but many of us would, I think, hesitate to apply this when seeing a case

SCHWARTZE MASTOIDECTOMY 50 CASES

OF CASES NEEDING OPERATION

31 HAD RECEIVED NO ANTIBIOTIC TREATMENT FROM THE GENERAL PRACTITIONER

13 HAD NOT RECEIVED ENOUGH ANTIBIOTIC TREATMENT

2 HAD RECEIVED THE WRONG ANTIBIOTIC

4 WERE ADMITTED FOR OPERATION FOR OTHER REASONS

Figure 5

SCHWARTZE MASTOIDECTOMY 50 CASES

AFTER OPERATION THE MEATUS WAS DRY IN 14 DAYS OR LESS IN 23 CASES

THE MEATUS WAS DRY IN 4 WEEKS IN 26 CASES

THERE WAS NO MEATAL DISCHARGE AFTER OPERATION IN 1 CASE

IN NO CASE DID OTORRHŒA PERSIST FOR LONGER THAN 2 MONTHS AFTER OPERATION

Figure 6

of otorrhea of six months' standing in a sclerotic mastoid. I think that antibiotics should be used in full doses for four weeks if there is any doubt, even when the mastoid is sclerotic.

III. More Serious Indications Such as Lateral Sinus Thrombosis and Meningitis.—These indications I would contest. Meningitis is now a rare complication of otitis media, and I think everyone would recommend antibiotics, given if necessary by lumbar puncture, rather than the old-fashioned incision of the mastoid process. Lateral sinus thrombosis is so rare that I have not seen a case as a complication of acute suppurative otitis media since 1945.

Facial paralysis is not mentioned by Ballenger but it might be discussed here, and I will give an example of my own. A child of 4 years was seen with a complete facial paralysis. He had suffered from an acute otitis media for three weeks, which had been treated with small doses of sulfonamide. The ear drum on the same side was thick, moist, and pale, and the hearing was very poor. A Schwartze mastoidectomy showed general infection of the mastoid. The face recovered in six weeks. I think this was the wrong treatment and I would prefer full doses of antibiotics for four weeks if I saw the child now, but I imagine there are some people who would not agree with me. Ten years ago, facial paralysis in the first week of an acute infection was seldom considered an indication for operation, and now facial paralysis, even when it occurs four or six weeks after the attack has begun, can well be treated by antibiotics alone.

Granulations or Sequestra of Mastoid Cells

Granulations at the edge of the drum membrane, or the evidence of dead and sequestrated mastoid cells (not an indication of Ballenger) have been considered as evidence to justify operation. That neither occurs in an early stage is true, but there is no reason to think that antibiotics will not produce a cure in these cases, as in other cases where antibiotics have not been given as soon as the infection occurs.

A patient was seen in October, 1951. She was 32 years of age, and complained of a discharge from the left ear for three weeks, with pain at the beginning for 24 hours. This was the first attack. She had not been treated by antibiotics. The ear showed a profuse discharge, which yielded a hemolytic Streptococcus on culture, sensitive to penicillin. The drum showed granulations at the posterior margin, and the hearing was much reduced. She was admitted to hospital and treated with penicillin, but there was no improvement after four days. A Schwartze mastoidectomy was advised, and the ear was dry and the drum healed seven days after operation.

This was my own case, and if I saw it now I would recommend a longer course of penicillin. I do not think the success of the mastoidectomy justified the operation.

Sequestra of mastoid air cells are unusual but I can only say that this occurred once in my experience when I was treating a child aged 4 years, and the patient recovered after seven days' treatment with penicillin, without operation.

Masking

When penicillin came into general use, there were many articles published warning against the danger of masking by penicillin. We have found, on the whole, that this has not been borne out by experience. Masking is rare when penicillin is used, and if improvement of the infection occurs with treatment it is likely to be maintained.

I have three records, however, which can be fairly considered as examples of masking. They are all cases of adults with a history of two or three weeks of otorrhea after an acute onset, in which antibiotics had not been used until I saw them. In each case penicillin dried the ear up completely in four or five days, but there was still pain and deafness, and the discharge

SCHWARTZE MASTOIDECTOMY 50 CASES

DEAF AFTER OPERATION

WITH A LOSS OF MORE THAN 30 DECIBELS FOR THE SPEECH FREQUENCIES 9 CASES.

Figure 7

recurred in 48 hours after treatment was suspended. All three cases recovered after mastoidectomy. In this connection I would suggest that deep-seated pain in the ear, and especially pain at night, is still significant as a sign of mastoiditis needing operation, even when antibiotics are properly used.

Deafness

There has, at several times, been a suggestion that acute otitis media will cause deafness unless a myringotomy is done, or unless mastoidectomy is undertaken promptly when it is called for.

This paper does not deal with myringotomy, but the general opinion everywhere seems to be moving against myringotomy in otitis media and tends to favor antibiotics alone. The argument that deafness occurs unless myringotomy is done is difficult to counter with figures, but general experience agrees that an ear may sometimes remain deaf even when antibiotics have produced a cure of the infection, and that in these cases myringotomy would not have saved the hearing.

In this connection it is disconcerting, even if it proves nothing, to find that a fair proportion of cases are deaf after mastoidectomy (Figs. 7 and 8). It may well be argued that only in the severer cases is operation undertaken, but at all events a Schwartze mastoidectomy cannot, from these figures at all events, be considered as a means of preventing deafness in otitis media.

Before passing to consider the organisms themselves and their response to antibiotics, it might be wise to consider one type of

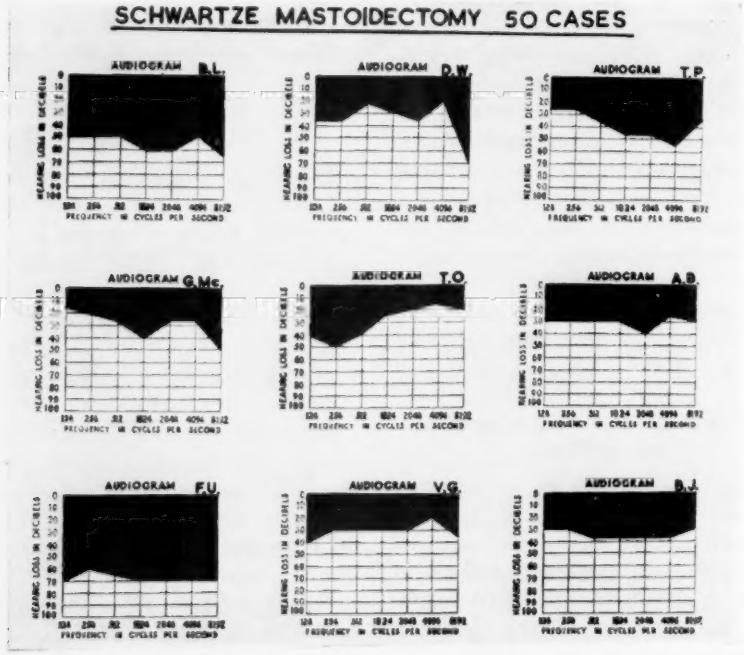

Figure 8

case which I have treated differently in recent months as a result of reflection over this paper. A patient who comes to the outpatient service with a discharging ear which has lasted for six months or even longer is not to be considered, in my opinion, as a true example of chronic otitis media, if the x-ray shows a cellular mastoid. There is a possibility that the infection may have begun as an acute otitis media, even if there had been no pain at the beginning, and antibiotics should therefore be given for several weeks before advising operation. That the same course of events may occur in a sclerotic mastoid has already been considered, but is less likely.

Lastly, we may speculate what will happen in future to the behavior of the bacteria themselves. In this, our experience in London is certainly less than in the United States, and I would only give one or two tentative suggestions. The first is that, on the whole, the resistant organisms are less virulent than those which are sensitive to antibiotics, so that resistance usually shows itself by a persistent otorrhea and not by a seriously ill patient. Secondly, there is no doubt that bacterial sensitivity to antibiotics is changing, although with us the change is slow and we still use penicillin in most cases, and only occasionally do we have to use tetracycline (Achromycin) or another antibiotic. On the other hand, I wonder what will be the comment on the subject of this paper in 20 years' time, or even in 10 years' time. The general opinion of pathologists is that general bacterial resistance to penicillin may develop quite soon and that this resistance may spread to all the other known antibiotics. For this reason, if not for any other, we should, as surgeons, remember the value of the Schwartze mastoidectomy.

My colleagues at the Royal National Throat, Nose and Ear Hospital gave me permission to examine their cases. Dr. T. V. L. Crichlow provided the x-ray reports and Dr. I. Friedmann the reports on the bacterial cultures. The diagram in Figure 1 is by Prof. F. C. Ormervel.

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Nystagmography During Counterrolling of the Eyes in Man

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Anyone who has reviewed the literature of the last 10 years on the various methods of nystagmography has no doubt been struck by the preference given to electronystagmography over other techniques.

Electronystagmography has many advantages. It is easy for both examined and subject. It does not entail a great deal of expense as long as there is an opportunity to make use of an existing electroencephalograph or electrocardiograph.

There are, however, a few shortcomings, especially when exact computations are needed.

The cornea-retina potential, the source of the electric energy of which the fluctuations are recorded, shows a quantitative correlation with light adaptation, according to a series of experiments by G. and J. ten Doesschate.[1] Consequently, experiments can be executed only in the dark, and even so calibration at regular intervals is necessary. The plane of calibration should be the plane of the nystagmus.

According to Lansberg,[2] the direction of gaze is an important factor. Generally speaking, the potential shifts are a sine function of the ocular excursions, and a cosine function of the angle between the direction of gaze and the plane of ocular rotation.

In case of nystagmus around the anatomical and electric axis of the eye (rotatory nystagmus) no electric signal will be elicited.

However valuable electronystagmography may be, especially for clinical purposes, in a number of instances it cannot meet all requirements.

The ideal nystagmograph must be capable of recording accurately not only the number of beats, the duration of a series and the angular velocity of the slow and quick phases, but also the amplitudes, the direction of the movements (horizontal, vertical, rotatory, or any combinations), and the position of the eyeball.

More than 25 years ago I had the opportunity of developing a method of optical nystagmography that has been used many times since. It has proved its value by meeting all requirements mentioned.

Particular need for such a method existed when it was necessary to investigate the movements of the eyes resulting from a combination of vestibular nystagmus and counterrolling of the eyes.

Details about the optical method of nystagmography, as used in this and other investigations, have been published elsewhere.[3-5] A few salient features will be related here.

1. Recordings are made on a moving strip of photosensitive paper in a camera.

2. A tiny pin of platinum is fixed on a little cap of black silk. Its form reminds one of a miniature umbrella that has been upturned by the

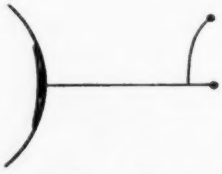

Fig. 1.—A tiny pin of platinum provided with two little reflecting knobs, one at its extremity, the other at a little side branch. The pin is fixed on a little cap of black silk, to be placed upon the cocainized cornea.

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Read before the Sixth International Congress of Otolaryngology, Washington, D. C., May 6, 1957.

wind. The silk cap has the same diameter and rounding as the cornea. The platinum pin is provided with two little reflecting knobs of platinum, one at its extremity, the other at a little side branch. Its total weight is 38 mg. (Fig. 1).

3. The two platinum knobs reflect the light from a tiny electric bulb into the camera, acting as pointers of the position of the eyeball either in rest or in motion.

4. The objective of the camera is provided with a so-called "reversing prism", its base placed at an angle of 45 degrees with the horizontal. A beam of light entering the objective is divided into two halves, one proceeding directly, the other passing through the prism which alters its course. By this procedure one reflecting knob is represented by two images on the film. Each image moves in a different plane and direction, making it possible to analyze the direction of the nystagmus, even if it coincides with the direction in which the film is moving. The addition of the second reflecting knob brings about four simultaneous curves. By comparing the angles of these curves it is possible to analyze any complicated movement of the eye, even a rotation around the sagittal axis.

5. By a special arrangement it is possible to make snapshots of the four dots of light while the eye and the film are at rest.

6. There is a time indicator on the film.

7. Optic stimuli during the experiment are excluded, the examined eye being covered by the little cap of black silk, the other eye being covered by a bandage.

8. A device has been constructed in order to be able to turn a subject around various axes and in various positions. Movements of the head in relation to the body or to the recording instrument are excluded.

By this method a great number of subjects have been examined while they were rotated around their longitudinal axis at various angular accelerations, both in a vertical and in a horizontal position.

The object was to study the influence of the phenomenon of counterrolling of the eyes * upon a vestibular nystagmus when both reactions occur simultaneously.

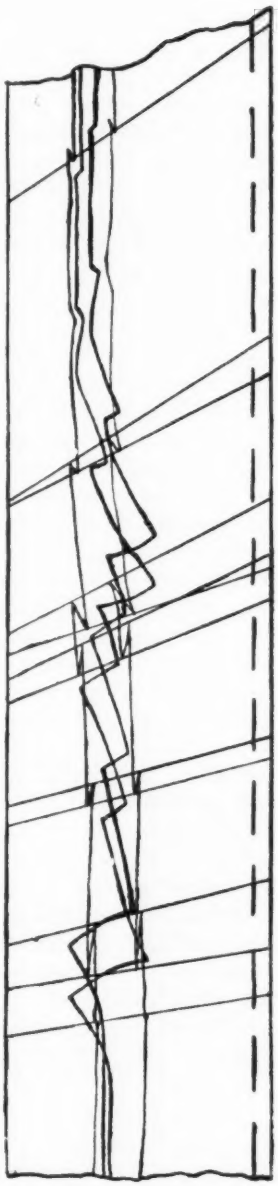

Fig. 2.—Series of nystagmus movements provoked by a rotating movement around a longitudinal axis in a horizontal position, from 60 degrees to the right, via the supine position, to 60 degrees to the left. In order to facilitate the analysis of the nystagmograms, lines have been drawn connecting identical points (the turning points of the curves). These lines indicate the position of the meridian of the eye coinciding with the sidebranch of the platinum pin. It is clearly evident that there is

(Legend continued on next page)

* Counterrolling (in German *Gegenrollung*, or *Raddrehung*) has been known for many years as a reflex for which the stimulus originates in the maculae of the sacculi by a shift in the direction of gravitation. If the head is moved slowly from a vertical position sidewards or from lying backward is turned on its side, the eye has a tendency to maintain its position in space by a rotating movement in the opposite direction in a frontal plane.

These combined ocular movements occur when a person is being rotated around his longitudinal axis, not in a vertical but in a horizontal position.

A comparison of the nystagmograms made in a vertical position with those made in a horizontal position reveals a marked difference. The former shows an "ordinary" perrotatory nystagmus; in the latter case the nystagmus is complicated by a gradual and oscillating movement of the vertical meridian of the eye, the counterrolling, which can be measured from the curves (Fig. 2).

By repeating the experiment (i. e., rotating the head around the longitudinal axis in a horizontal position) very slowly, in a subliminal acceleration, the same counterrolling can be observed; however, without nystagmus. It is recorded at various stages by making instantaneous photographs of the four dots of light with the camera.

From these observations it has been proved that the angle of counterrolling, with or without combined nystagmus, is the same. It varies between 10 degrees and 20 degrees.

A review of the literature on the clinical methods of examination of the vestibular organ gives the impression that most authors are concerned about the reactions of the semicircular canals.

The possibility of recording otolithic reflexes with or without simultaneous cupular reactions may prove to be a stimulant to further research in the field of vestibular physiology and pathology.

a gradual displacement of the vertical meridian of the eye during the nystagmus movements (in total ±20 degrees). The interrupted line along the edge of the film indicates the time (five interruptions in two seconds).

Summary

However valuable electronystagmography may be, in a number of instances it cannot meet all requirements, especially when the eye is moving around its sagittal axis, (rotatory or complicated nystagmus with a rotatory component). A description is given of a method of optical nystagmography, as I have used it, with the object of studying the influence of the phenomenon of counterrolling of the eyes on vestibular nystagmus when both reactions are brought about simultaneously by rotating a subject around his longitudinal axis in a horizontal position.

19 Surinamestraat.

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Editorial Comment

In the paper, "New Middle Ear Mechanisms for Normal Hearing," * Dr. Samuel Rosen presents case reports showing remarkable postoperative audiograms in situations where the entire ossicular chain has been disrupted and a fenestra placed in the footplate without continuity with ossicles or eardrum. He states that his findings seriously challenge the Helmholtz theory of hearing and the many observations of recent physiologists. In cases with an intact drum, a closed middle ear, and no ossicular continuity, Rosen is nevertheless able to demonstrate normal hearing through the function of an open fenestra in the footplate of the stapes.

These observations are, of course, disturbing to all currently accepted theories of middle ear function. It is difficult to reconcile these reports with other observations by the same author in which he shows equally excellent results where ossicular continuity was maintained. One wonders as to the fate of the average 26 db. contributed by the impedance matching mechanism of the middle ear as a transducer or acoustic "transformer." This normal 26 db. contribution of the middle ear has been demonstrated time and time again in experimental studies and in clinical observations involving stapes surgery, fenestration surgery, and tympanoplasty.

Furthermore, it is not clear as to what fate befalls the opening in the footplate from which undoubtedly a large amount of perilymph must be lost, since no mention is made of any covering to this footplate fenestra. One also wonders as to the fate of the utricle and saccule, which could very easily be disturbed by the type of instrument used in producing a footplate fenestra. Some otologic surgeons have observed cerebrospinal fluid leaks following footplate instrumentation of even less magnitude.

This paper deserves serious study and is challenging both surgically and physiologically, but it must be evaluated with care. It would thus seem to be unwise for this procedure to be adopted in its presently described form.

Stapes mobilization has been a very important contribution to otologic surgery in its present renaissance, with restoration of hearing to many thousands of people, but there have been some significant instances of postoperative complications. Caution is indicated in the evolution of the stapes mobilization approach but extra caution must be urged in the rather daring procedure described in this paper.

Although clinical observations may be very stimulating to physiologic theories, the ultimate solution to physiological problems requires methodical analysis and carefully planned experimental studies. No firm theory should be challenged without adequate quantitative experimental data.

* This issue, page 428.

New Middle Ear Mechanisms for Normal Hearing

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In 1863 Helmholtz [1] formulated the idea that the middle ear apparatus—that is, the tympanic membrane and ossicular chain—serves as a sound transformer. Drum vibrations are transmitted along the ossicular chain in a way that reinforces the alternating pressure of the sound waves. This increased pressure is then transmitted to the cochlear fluid by the piston-like action of the stapedial footplate moving the fluid back and forth.

For 100 years, this lever-like mechanical transformer action of the ossicular chain has been accepted as the necessary condition for normal hearing, because loss of the transformer action by interruption or damage of the ossicular chain by disease or accident causes a serious loss of hearing, 30 db. Békésy [2] studied five ears in which the drum, malleus, and incus were all absent. He reported an average loss in these cases of 15 to 65 db., which varied with the frequency. It is also of interest that there is a residual loss of about 30 db. below normal following the Lempert fenestra nov-ovalis operation, presumably because the transformer action of the middle ear is lost.

Similar loss of hearing has been found by measurements of electric potentials of the cochlea in experimental animals when the middle ear transformer mechanism is impaired. Wever, Lawrence, and Smith [3] have demonstrated in the cat that separation of the incudostapedial articulation, with all other parts of the middle ear intact, produced an average loss of 52 db. They explain this extreme loss in two ways: (1) the remaining drum, malleus, and incus now reflect and absorb sound and are therefore worse than useless, and (2) sound has equal access to both windows, which disturbs the delicate and crucial phase difference.* When

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* In some cases of otosclerosis, as soon as the fenestra ovalis is made, the hearing improves only to diminish when the drum is replaced. Creation of an opening in the drum posterosuperiorly usually causes the hearing to improve immediately.

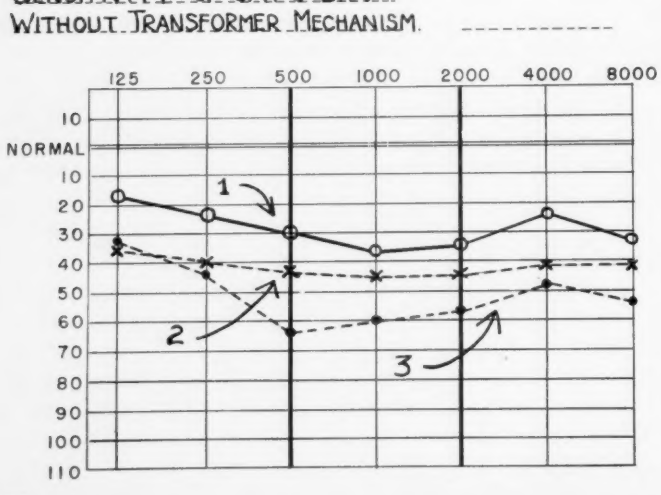

Fig. 1.—Hearing loss in cat under three conditions of interruption of ossicular chain with consequent loss of transformer action: (1) when sounds are delivered exclusively to the oval window=28 db. loss; (2) when sounds are presented through the meatus with drum, malleus, and incus removed=42 db., and (3) sounds presented where only the incudostapedial articulation was separated, all other parts being in position=52 db. loss. (Data from Wever, Lawrence, and Smith.[3])

the drum, malleus, and incus are removed, the hearing loss is about 42 db., but when the sound is directed exclusively to the stapedial footplate, the average loss is only 28 db.

The present report proposes to present evidence that (1) the normal ear in man can continue to hear normally after the incudostapedial articulation has been widely and irreversibly separated, thereby interrupting the middle ear transformer mechanism, and (2) that normal hearing can be achieved and maintained after Rosen's fenestra ovalis operation [4,5] through the rigid and immobilizable footplate. In some of the latter cases, the very resistant crura were not fractured, while in others the crura were fractured and were removed along with the incus for better visualization of the entire naked stapedial footplate.

I. Continuance of Normal Hearing after Wide Separation of the Incudostapedial Articulation

A woman 29 years of age suffered from severe attacks of bilateral Ménière's disease for several years. In repeated tests, the hearing in both ears was always found to be normal. To relieve the Ménière's attacks a bilateral chorda tympani nerve section [6,7] was performed through the ear canal under local anesthesia on March 17, 1954. No bone of the posterior canal wall was removed, and only the edge of the incudostapedial junction could be seen. During the operation on the right ear the long process of the incus was inadvertently hooked outward for a short distance toward the operator, along with the chorda tympani nerve. The dislocation of the stapedial end of the long process of the incus was so wide that it almost touched the middle of the hammer handle (Fig. 2). The incus became rigidly locked in the incudomalleolar articulation as if in a vise and could not be moved from this abnormal position. The disconnected articular surfaces of the incus and stapes were separated by a space of at least 6 mm. The drum was replaced and was not near the head of the stapes.

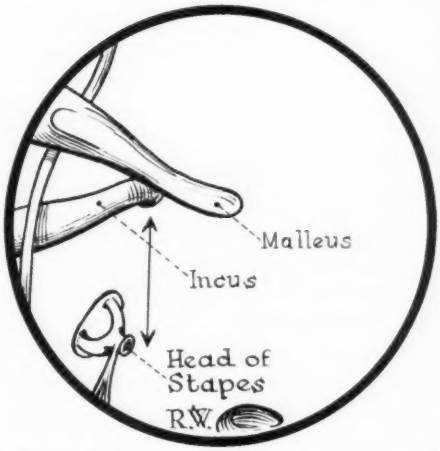

Fig. 2.—Schematic drawing of the wide and irreversible disruption of the incudostapedial articulation in an ear with normal hearing. (See Fig. 3, audiogram of this case three and a half years later with the same position of ossicles.)

In the three and a half years since the operation, this abnormal position of the long process of the incus has not changed and can still be seen through the normal drum. The drum is in normal position, is not scarred or retracted by any adhesions, and is not in contact with the head of the stapes. Repeated audiological tests over the past three and one-half years show the same normal hearing in both ears as before the operation † (Fig. 3). This would indicate that the architecture of the middle ear transformer mechanism described by Helmholtz is a sufficient condition for normal hearing but not a necessary one.

II. Normal Hearing Without the Middle Ear Transformer Mechanism After the Rosen Fenestra Ovalis Operation

Rosen has shown [4] that normal and near-normal hearing has resulted in ears where the transformer mechanism is interrupted by creating a fenestra through the rigid footplate into the vestibule. This hearing improvement has been maintained for periods up to 21 months at the last testing.

† Since section of both chorda tympani nerves, all attacks of vertigo have vanished.

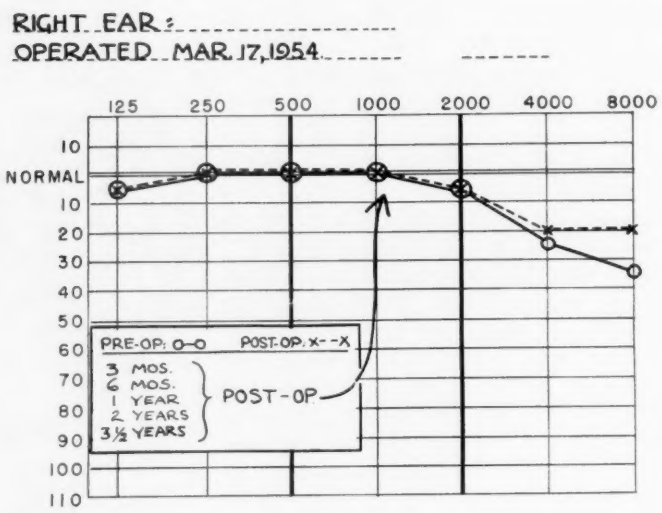

Fig. 3.—Pre- and post-operative audiogram, showing normal hearing maintained three and a half years after wide and irreversible separation of the incudostapedial articulation.

Case 1.—A 41-year-old woman had bilateral progressive deafness and tinnitus for seven years. In May, 1954, a mobilization of stapes was performed on the left ear at the stapedial neck (indirect method). The hearing improved from the preoperative level of 40 db. loss to 15 db. loss for pure-tone and speech reception. On Dec. 2, 1954, a stapes mobilization was performed on the other ear (right) at the stapedial neck. The hearing improved from a preoperative loss of 35 db. to 20 db., for pure tone and speech and then regressed after eight months to the preoperative level. On Sept. 12, 1956, this patient's right ear was reoperated on. The crura seemed to have been slightly fractured at the initial mobilization operation. Gentle manipulation of the crura now showed preternatural mobility, indicating fracture. The fractured crura were removed. The fenestra ovalis was made through the body of the footplate with the hooked fenestrator. The hearing improved on the operating table. Two weeks later the speech reception threshold was at the 20 db. level. Seven months after fenestration of the oval window on the right ear, the hearing was normal and it has been maintained to date (Figs. 4 and 5).

Case 2.—A 38-year-old man had bilateral progressive deafness for nearly five years. His preoperative loss for pure tone and speech was 30 db. in the right ear and 45 db. in the left. On Feb. 27, 1957, the right stapes was found to be extremely

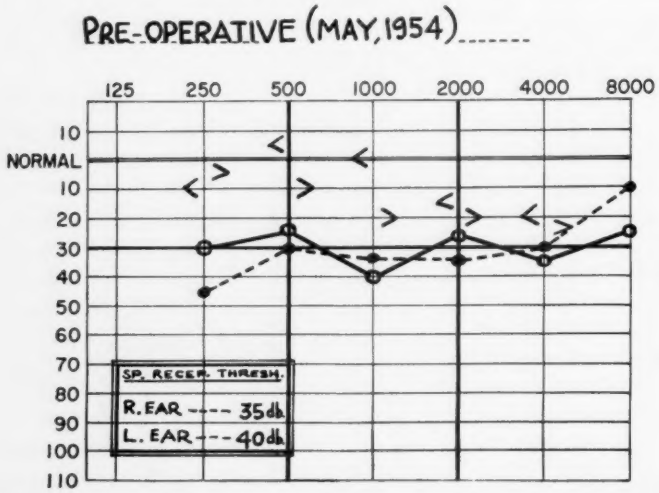

Fig. 4. — Preoperative audiogram of a 41-year-old woman (Case 1) who had bilateral progressive deafness for seven years.

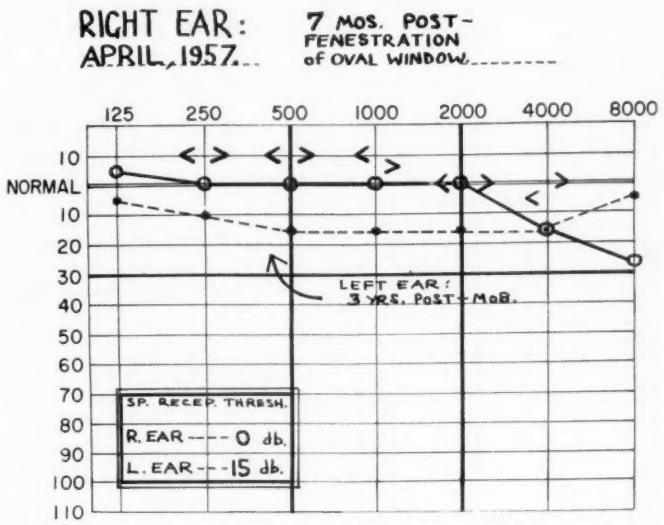

Fig. 5.—Normal hearing after fenestra ovalis in right ear. Fractured crura excised. Near-normal hearing in left ear three years after mobilization at neck of stapes.

rigid and immobilizable. The crura remained intact, but the incus was removed to obtain a better view of the footplate. A fenestra ovalis was then created through the rigid footplate. The hearing improved on the operating table. The stapedial footplate and the entire stapes was as rigid and immobilizable after fenestration of the oval window as before. Five months after operation, the patient's hearing was 5 db. above zero by pure-tone and speech audiometry (Fig. 6).

CASE 3.—A 41-year-old woman had bilateral progressive deafness and intermittent tinnitus for nine years. The preoperative hearing loss for pure tone and speech was 45 db. in the right ear and 50 db. in the left. On Nov. 3, 1955, the right stapes was found to be extremely rigid and immobilizable. The crura remained intact. A fenestra ovalis was created at the periphery of the rigid footplate at its inferior aspect. The hearing improved to the 15 db. level for pure tone and speech and has been maintained to date.

On Sept. 27, 1956, the other ear (left) was operated on. The rigid stapes was immobilizable although the crura did not fracture. A fenestra ovalis was created through the body of the rigid footplate, and the hearing improved on the operat-

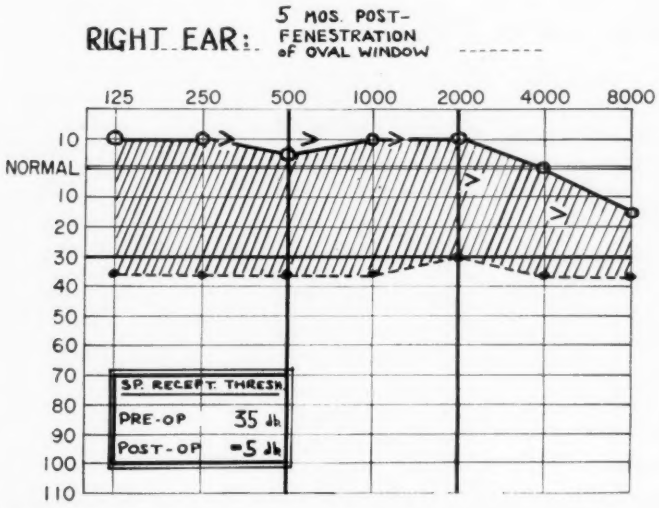

Fig. 6.—Normal hearing after fenestra ovalis through the immobilizable footplate. Crura remained intact, but incus was excised to permit full visualization of rigid footplate. The middle ear transformer mechanism in this case was destroyed.

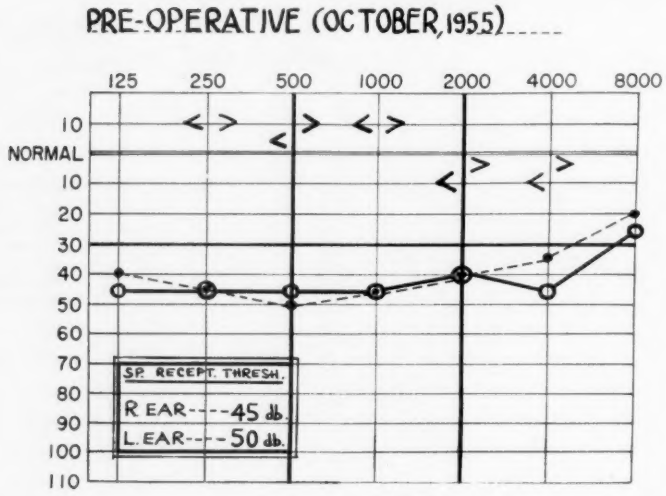

Fig. 7. — Preoperative audiogram of a 41-year-old woman with bilateral progressive deafness for nine years.

ing table. Two weeks after operation, the hearing was at the 25 db. level for pure tone and speech and continued to improve. Three months after operation, the hearing reached the 15 db. level, where it has been maintained to date. This patient now has near-normal hearing in both ears, as shown in the accompanying audiograms (Figs. 7 and 8).

CASE 4.—A 42-year-old woman had bilateral progressive deafness and intermittent tinnitus for two years. Her hearing loss in the right ear was 45 db. for pure tone and speech and 35 db. in the left ear.

On Sept. 11, 1956, the right stapes was found to be extremely rigid and immobilizable. The crura fractured and were excised along with the incus for better visualization of the entire footplate. A fenestra ovalis was then made by penetrating the body of the rigid footplate. Two weeks after operation the hearing was at the 25 db. level for speech. It is interesting to note the gradual improvement of hearing over a period of months in this case, as shown in the accompanying audiogram. At one and a half to three months postoperatively, the average loss by pure tone and speech audiometry was 10 db. In April, 1957, seven months after operation, her hearing was at 0 db. for pure tone and speech and has been maintained to date (Fig. 9).

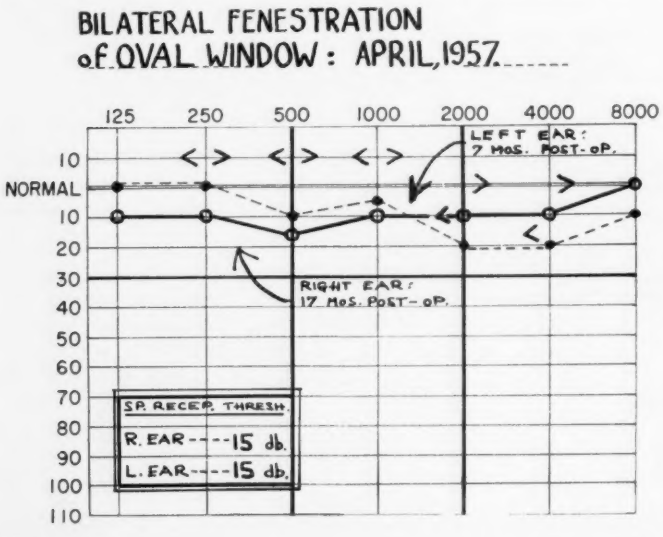

Fig. 8. — Near-normal hearing in both ears after bilateral fenestration of the rigid, immobilizable footplate. The ossicular chain remained intact in this case.

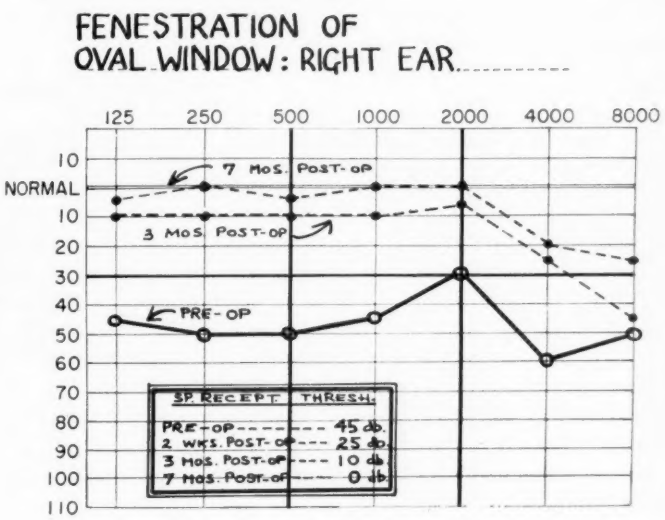

Fig. 9.—Normal hearing following fenestra ovalis. In this case the incus and fractured crura were removed for better visualization of the entire footplate. Audiogram shows progression of hearing improvement over a seven-month period.

We now have evidence that normal hearing can be maintained in man when the sound transformer mechanism of the middle ear is not present, i. e., when the incudostapedial articulation is broken and widely separated, and when a fenestra is made in the oval window through the immobilizable footplate, whether the incus and fractured crura are excised or not. The preservation of normal hearing in a normally hearing ear despite the interruption or separation of the ossicular chain would seem to challenge Helmholtz' concept of the middle ear transformer mechanism. Although quantitative analysis in experimental animals of the facts presented here is not yet available, the fact remains that normal hearing has been maintained without a middle ear transformer mechanism.

There appears to be, therefore, at least two different structural mechanisms capable of producing normal hearing: one with the transformer mechanism of Helmholtz intact and one without the middle ear transformer mechanism. These may be regarded only as known parts of a total phenomenon. In searching for new mechanisms for normal hearing we need not abandon the old and we may gain substantially from the new. It would be erroneous to disregard new facts. New problems must be faced and solved.

Summary and Conclusions

Normal hearing is made possible by the middle ear transformer mechanism. This is the concept of Helmholtz.

Normal hearing can be maintained in a normally hearing ear when the middle ear transformer mechanism is lost after the incudostapedial articulation is permanently broken; and (2) normal hearing can result from a fenestration of the oval window through the rigid, immobilizable footplate in otosclerosis.

New problems arising from the facts presented must now be faced and solved.

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Severe Epistaxis

Recent Advances in Its Treatment

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In 1942 I described the type of nasal hemorrhage associated with hypertension and arteriosclerosis,[1] and it was asserted that this variety of nasal hemorrhage seemed to be a distinct clinical entity.

In 1949 cardiovascular epistaxis was again described.[2] Its status as a distinct clinical entity was reasserted, and 28 cases were reviewed. In this latter article, the existence of a small group or plexus of blood vessels, lying at the junction of the nasal chamber and the nasopharynx, in the lateral wall, just beneath the inferior turbinate, was described. This group of vessels was given the name of the naso-nasopharyngeal plexus because of its location partly in the nose and partly in the nasopharynx. It was pointed out that this plexus was often the site of bleeding in cardiovascular epistaxis. In both of these articles a method of precision or spot packing was described which was found to be useful in many of these cases.

While it is still my belief that severe spontaneous epistaxis, in the adult, is usually a manifestation of cardiovascular disease, I feel that in some of these cases another factor may be involved. If there is another factor, it is apparently unknown up to the present time. It seems strange to me that two apparently healthy women approximately 50 years of age, in whom the blood pressure was not unduly elevated, should suddenly begin to bleed freely from the choanal area of the nose. It is easy to say, of course, that some vessel had been weakened by vascular disease, presumably arteriosclerosis, and so ruptured, even from a moderate increase in the blood pressure.

However, we have no proof of this, and one may wonder if there was not some preexisting defect or weakness in a vessel, perhaps congenital in origin. Sudden cerebral hemorrhages have been found to be due to the rupture of congenital aneurysms, and perhaps the same condition may exist in the nasal chambers. There have been theories advanced to explain various types of hemorrhage, perhaps the best known being that of Jacobson,[3-6] who ascribes many cases of epistaxis, as well as hemorrhages in other localities, to endocrine imbalance, usually a lowering of the estrogen content of the blood. Up to the present there is no real proof of this, even though it has led to therapy with estrogens administered intravenously, under the name of conjugated estrogenic substances (Premarin), which do appear to be effective in some cases of epistaxis. For this reason it seems as if the whole question of epistaxis should be reexamined from every conceivable view point in order that our treatment may be made more effective and less troublesome to the patient than it has been in the past. Medicine has made so many real advances in recent years that people have come to expect effective treatment with a minimum of discomfort. Some of the procedures still used to arrest epistaxis appear to be distressing enough to make the patient question our competence in this field. Some advances have recently been made in the treatment of epistaxis, but more are still needed, and I feel that the suggested reexamination of the whole subject may produce a solid foundation for much further improvement.

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Where do we stand today in the treatment of severe epistaxis?

In the past there have been, in the main, three methods used in the permanent arrest of severe nasal hemorrhage. They are, first, cauterization, both chemical and physical; second, packing or tamponage, and third, vascular ligation.

Coagulation is included under physical cauterization because I feel that its end result is the same. I do not believe that chemical cauterization is effective in active bleeding from vessels of any considerable size. The actual cautery or coagulation applied over and adjacent to a break in a vessel stops bleeding by the formation of eschars which effectively seal the open ends while the slow process of healing takes place. Packing is a form of pressure applied either over the site of hemorrhage or somewhere over the proximal portion of the bleeding vessel. Occasionally it may be necessary to pack distally to the site also, especially where the break is close to the anastomosis between branches of the sphenopalatine and ethmoidal vessels. Obviously, to effectively compress vascular trunks the pressure must be quite firm, probably more so than when the pack is placed directly over the break in the vessel. When the site of bleeding is at the choanal end of the inferior meatus, a choanal plug drawn into place from the pharynx may be successful. Also with a tight choanal plug in place anterior packing may be applied more effectively. A good method of making and applying a choanal pack has been described by Dickie[7] and advocated by Hallberg.[8]

Dickie describes the making of a posterior plug as follows:

> To put in a posterior plug, proceed as follows: A strip of gauze about 2½ to 3 inches wide is folded lengthwise, one inch from one edge. It is then rolled up tightly so that one side is much thicker than the other.
>
> When the thick end is about ¾ inch thick, cut off the surplus and bind tightly round the middle with string, leaving the ends long. Wind the ends crisscross round the thinner end and tie tightly. One end of the string may be cut off about 4 or 5 inches and the other left long. The roll is now wedge-shaped with the strings attached to the narrow end. A rubber catheter or Bellocq's sound is passed through the nose and fished out of the mouth. The long end of the string is tied to the catheter which will pull the plug into the back of the nose with a finger in the mouth to hold the palate forward. Once in the posterior naris, it is pressed firmly home from behind, aided by traction from in front.
>
> To keep the plug in place take a section of rubber tubing of large diameter (½ inch or more) and place it across the nostril and wind the string around it crisscross. Better still, transfix it with a large needle and draw the string through and wind round it. This will maintain a certain amount of pressure without damaging the alae.
>
> The tampon will stay in place without requiring to be disturbed for a week or more. If necessary the front of the nose can be packed with ribbon gauze as well. The other end of the string is allowed to hang down behind the palate to where it is just visible. It is useful for extracting the plug. If this pack is well placed it will not interfere with the eustachian tube nor cause otitis media.

When packing is used successfully it has to be left in place long enough for some sort of repair to take place or the bleeding will recur on its removal. I have arbitrarily left packs in place for five days, and, if no bleeding occurred, removal of the pack was accomplished without recurrence.

If you have a patient whom you do not want to pack, and I think many of us would like to avoid packing if possible, you may try the medical treatment. The most widely used agent apparently is a mixture of conjugated estrogens (Premarin), developed a few years ago for intravenous use. Another one is carbazochrome (Adrenosem), a derivative of epinephrine (Adrenalin), given either intramuscularly or by mouth. Either of these may be given quite freely. Neither appears to have any serious toxic effects in the dosage used. Twenty milligrams of conjugated estrogenic substances may be given every two hours and then reduced in frequency as the bleeding is controlled. Carbazochrome may be given intramuscularly in 5 cc. doses every hour for 24 hours and then the frequency is decreased. Vitamin K does not seem to be helpful unless there is a prolonged prothrombin time.

Peele [11] has reported 23 cases of epistaxis in which carbazochrome was successful in controlling the bleeding and Menger [12] has reported five cases which were successfully treated with conjugated estrogenic substances.

When all of the preceding methods prove insufficient to keep the bleeding stopped, ligation of one or more arteries will have to be carried out. I feel that in the type of epistaxis under discussion the sphenopalatine artery or one of its branches is the bleeding vessel in the great majority of cases. In some traumatic hemorrhages the anterior ethmoidal artery may be more often involved. If the site of bleeding is localized to the inferior and lower two-thirds of the nose, the external carotid may be ligated in the neck, the internal maxillary may be ligated behind the antrum through a Caldwell-Luc approach, or the sphenopalatine may be ligated soon after it enters the nose, the approach being through a frontoethmoid sinus incision. In the latter case the anterior ethmoid artery is ligated first, against the orbitonasal wall. The ethmoid cells are then exenterated, exposing the anteroinferior wall of the sphenoid sinus preparatory to the ligation. Silverblatt [10] is a very strong advocate of the latter procedure. He feels that ligation of the sphenopalatine is much more apt to permanently arrest the hemorrhage than is ligation of the external carotid. In some cases it has been necessary to ligate the anterior ethmoid artery after ligation of the external carotid failed, and in others it has been necessary to ligate both external carotids when ligation of one was ineffective. In my practice the external carotid has been ligated six times over the last 20 years, and in each instance it was successful, though in two or three cases there was slight bleeding at the end of about 24 hours. Hallberg, who perhaps has had as extensive experience with vascular ligations for nasal hemorrhage as anyone, has usually preferred to ligate the external carotid artery.

The surgical aspect of the subject has been very ably reviewed by Silverblatt,[10] who has given us some new and definitely provocative ideas in the surgical approach to the problem. I believe all of us should read his paper and be in a position to adopt or reject the procedures he advocates so vigorously. During the last 18 months there have been five cases in my practice which have been classified as severe epistaxis.

The first of these was a male Negro, 52 years of age, who complained of intermittent nosebleed of three days' duration. After removal of the clots of old blood there was no active bleeding, and the site of origin could not be definitely located. A suspicious area on the left inferior turbinate was cauterized, and he was allowed to go home. For the next five days he appeared in my office, each time complaining of some bleeding, but at each examination his nose was found to be dry. Then, after an interval of two days he complained of a severe hemorrhage, though it had again stopped when I examined him. He was given 20 mgm. of conjugated estrogenic substances intravenously and had no further bleeding. The conjugated estrogenic substances were repeated in 48 hours.

The second case was that of a 49-year-old housewife, apparently in good health, who began to bleed suddenly on a Sunday afternoon. Examination at the office showed she was still bleeding, but upon removal of the clots and old blood, the bleeding ceased. The site of hemorrhage was not found, but because it was felt the bleeding was almost sure to recur, she was sent to the hospital. She was treated with intravenous conjugated estrogenic substances and intramuscular carbazochrome. There were several episodes of bleeding during the next 30 hours, but they gradually lessened in frequency and amount and then stopped. She was discharged from the hospital four days after admission. There has been no recurrence.

The third case was that of a 53-year-old woman who was sent to the hospital for a nosebleed which recurred several times. In spite of the free use of conjugated estrogenic substances and carbazochrome and choanal packing, the bleeding could not be controlled. Several transfusions were given, and for a time it appeared that the bleeding was being brought under control, but on the fifth hospital day after a particularly severe hemorrhage, she was taken to surgery at 11:00 p. m., when the external carotid was ligated under local anesthesia while another 500 cc. of whole blood was given. From this time her recovery was uneventful except for a minor episode of bleeding 24 hours later. She was discharged 11 days after admission and one week after the ligation.

The fourth patient was a 67-year-old man with choanal and anterior packs in place bilaterally.

These were introduced in the emergency room by the resident the night before, and two ampules of carbazochrome were given during the night. The next morning he was bleeding some and he was very apprehensive. He was given reassurance and treatment with conjugated estrogenic substances was begun, 20 mg., q. i. d., intravenously. The bleeding soon subsided, and the anterior packs were removed. He made good progress until he got out of bed two days later, when he started to bleed again. This soon stopped. The next day the choanal plugs were removed, and he was discharged, 12 days after admission.

The fifth patient was a 74-year-old woman who had definite hypertension. She began to bleed suddenly one evening from the left nostril. Office examination at 1:30 a. m. disclosed a small spurting hemorrhage in the anterior part of the left inferior meatus up under the inferior turbinate. It was inaccessible to coagulation, so a spot pack of cotton in peroxide was wedged into place supported by two more packs. This arrested the bleeding, but she was sent to the hospital and given conjugated estrogenic substances intravenously twice a day for 48 hours. She was discharged on the third hospital day after removal of one of the nasal tampons. The night after she arrived home she had a small amount of bleeding. The packs were removed 36 hours later, and she appeared to be all right. However, two days later she began to bleed again, and, as intermittent bleeding continued, spot packing was again carried out in my office more completely and firmly than the first time. The packs were removed on the fifth and sixth days, and bleeding so far has not recurred.

This small series of cases contains one which resisted all forms of treatment until the external carotid artery was ligated. There are two cases which received medical treatment only, one conjugated estrogenic substances alone, and one conjugated estrogenic substances and carbazochrome. There was one in which bilateral choanal and anterior packs did not completely control the hemorrhage, but after the addition of conjugated estrogenic substances the bleeding ceased. I am inclined to give most of the credit to the conjugated estrogenic substances in this case, because the anterior packs were left in place only 18 hours and the choanal plugs about 30 hours. In the last case, I feel that the spot packing when it was properly applied, controlled the bleeding, and it was probably not necessary to give the carbazochrome.

I realize that this series is too small to mean very much, but the experience is mine and I am bound to receive certain impressions from it. My beliefs are, therefore, based on these cases, on past experience, and on reports in the literature. My present belief is that these substances, conjugated estrogenic substances and carbazochrome are of some value in the treatment of severe epistaxis. I regard them as definite aids in the management of these cases.

Conclusions

Our methods for the control of severe epistaxis, though usually successful have not been very efficient.

With the increase in longevity these cases will probably become more numerous.

Some definite improvement has been made in the management, but further improvement is needed.

As a basis for this improvement the whole subject should be reexamined, beginning with the basic factors, such as the vascular anatomy, especially as regards anastomoses in various areas of the nasal chambers.

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Audiology Section

JOSEPH SATALOFF, ASSOCIATE EDITOR

Auditory Tests in Surgery of the Stapes

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Tests of hearing are now being used in stapes surgery for (1) the selection of cases; (2) the setting of functional goals for each case operated on; (3) the prediction of results, and (4) the evaluation and reporting of results. In addition, hearing tests have been employed as a guide for the surgeon during the operation.

It is the purpose of this paper to deal with the application of hearing tests to the selection of cases.

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Air- and Bone-Conduction Thresholds

It is generally accepted that the relationship between the pure-tone thresholds of hearing by air conduction and by bone conduction, the so-called air-bone gap, is the audiologic basis of case selection for the surgery of otosclerosis. A normal or near-normal threshold by bone conduction in the presence of a significant air-conduction loss is believed to represent the most suitable condition for surgery. Conversely, cases in which the air- and bone-conduction thresholds show about equal loss are considered unsuitable. Since the results achieved by stapes surgery have demonstrated that the air-bone gap can be completely eliminated in successful cases, it has become apparent that any significant air-bone difference in-

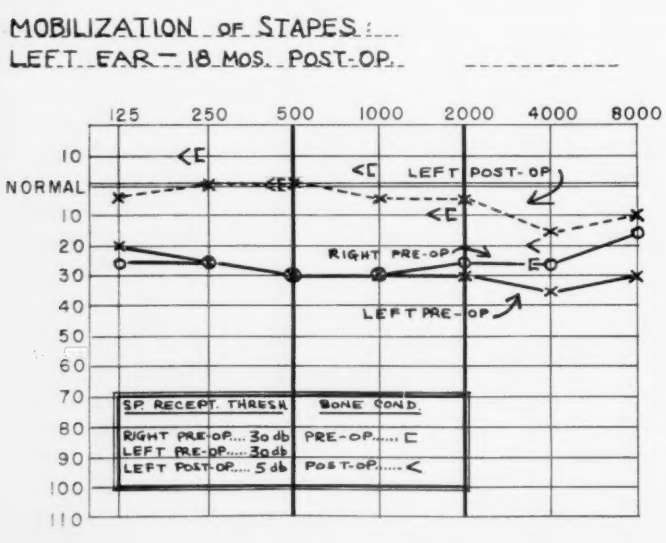

Fig. 1.—Pre- and post-operative audiogram. This patient applied for surgical relief of her deafness of 10 years' duration. It is interesting to note that while the hearing loss was only 30 db. bilaterally, the level which is often referred to as "socially adequate," this patient, as many others, apparently did not consider it so. Since mobilization of the stapes, the hearing in the ear operated on is now within normal limits. This audiogram was made 18 months postoperatively.

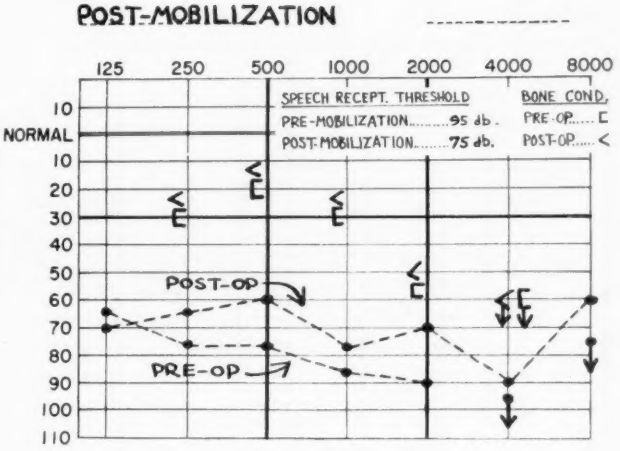

Fig. 2.—Postmobilization audiogram. The pure-tone average rose from 83 db. loss to 68 db. loss, and the speech reception threshold rose from 95 db. to 75 db. The patient can now use her hearing aid with greater usefulness.

dicates a possible case for surgery. A rule of thumb for a significant air-bone gap would be any over 15 db. in the speech range.

The Air-Conduction Threshold

Any patient who has sufficient loss by air conduction to cause him to seek corrective help may be a candidate for surgery. In general, the amount of loss does not enter critically into the decision to operate. Experience has demonstrated that losses as little as 25 or 30 db. and as great as 95 db. have yielded to this type of surgery for gains of 15 to 45 db. Figures 1 and 2 illustrate such cases.

The poorer ear may be selected for surgery, if (*a*) it is reasonable to expect that a successful result will increase the hearing beyond that of the ear not operated on, for an over-all improvement in hearing, or (*b*) a successful result promises to improve the hearing enough to provide the advantages of binaural hearing for soft or even for average conversational levels.

Prediction of over-all improvement may at present be based upon a knowledge of the

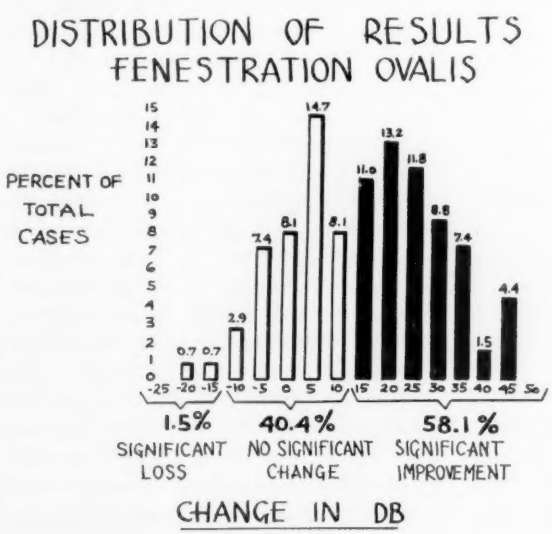

Fig. 3.—An example of a distribution chart, used in the prediction of results.

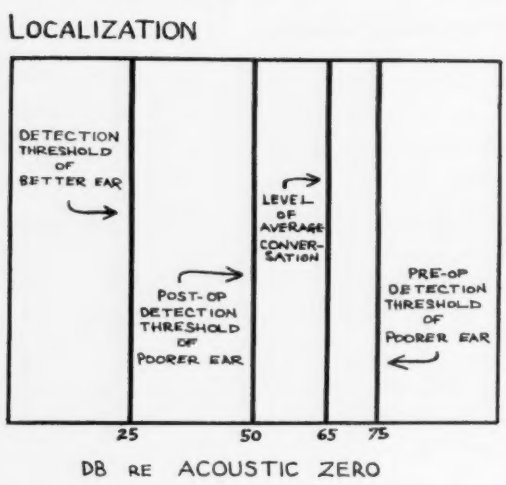

Fig. 4.—The detection thresholds are shown both preoperatively and postoperatively. The levels of the poorer, treated, ear reveal that this ear was not contributing to binaural hearing until after successful surgery of the stapes.

previous results. A distribution bar graph of results obtained by the surgery can provide a useful basis for such prediction. An example of such a distribution chart is given in Figure 3. This shows the results obtained in 136 cases of fenestration of the oval window as reported by Rosen at the Sixth International Congress of Otolaryngology.[1] Similar charts are made for each stapes procedure, such as mobilization at the neck and mobilization at the footplate.

In the matter of binaural hearing, Bergman has shown that the advantages of such hearing can occur for all sounds heard even slightly by the poorer ear.[2] Therefore, if a successful operation can improve the hearing of the poorer ear so that it can hear soft or average speech, binaural hearing will result for most situations. Figure 4 shows that before a stapes mobilization, the patient's poorer ear was unable to contribute sufficiently to effect binaural hearing except for sounds of unusually high intensities. Postoperatively this patient has effective binaural hearing for sounds and speech at average conversational intensities.

The Bone-Conduction Threshold

The weaknesses of testing by bone conduction are well known. Differences in bone oscillators and in calibration from one audiometer to another, coupled with the variations in such aspects as placement of the oscillator on the skull, the amount of pressure applied, the thickness of the skin over the mastoid, and the impedance of each skull, are only part of the problem. Alteration of the middle ear by pathological processes also apparently contributes additional variables not too well understood as yet. To add to the confusion, the use or nonuse of masking in the nontested ear, and indeed the very presence of the masking phone itself on that ear, has been found to cause threshold shifts in the ear being tested. The major limitation in case selection imposed by these problems of bone-conduction audiometry relates to those cases in which the hearing in the two ears is markedly different. Here it is often difficult, if not impossible, to be certain about the bone-conduction readings for each ear. In such cases it is desirable to perform other tests which can give evidence about the function of the inner ear mechanism of the ear being considered for surgery. If there is no clear evidence of inner ear deafness, the problem of conductive versus inner ear lesion remains unresolved. It is generally felt, however, that if there is positive evidence that inner ear involvement is present, the case is unsuitable for surgery. This is not always true. Successful operative results have been achieved in cases of mixed deafness in which a con-

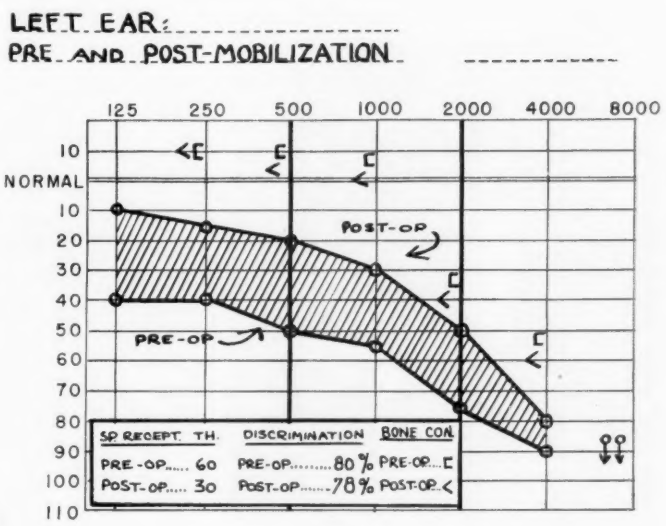

Fig. 5.—The poor hearing by bone conduction at 2000 and 4000 cps, together with the relatively poor preoperative discrimination score, did not prevent the significant improvement in hearing by stapes surgery.

siderable amount of inner ear deafness existed. It must be recognized, however, that for poorer bone-conduction thresholds the chances of success diminish. This is the meaning of the lower incidence of successes among "B" and "C" cases than in the "A" cases, as previously reported.

Discrimination Scores

Since the application of the discrimination test for the selection of cases for middle ear surgery, it has been claimed that patients showing subnormal discrimination were poor candidates. Our records seem to confirm this oftener than not. Nevertheless, one cannot ignore the fact that it is possible to obtain considerable hearing improvement even in cases with markedly inferior ability to discriminate, as shown in the case illustrated by Figure 5.

In this case the stapedial footplate was found to be moderately fixed. The fragile crura fractured immediately. Mobilization of the footplate was followed by hearing improvement from an SRT of 60 db. to 30 db., but the poor discrimination score (80% preoperatively) remained poor postoperatively (78%). This is reasonable since the discrimination score is a function of the inner ear rather than the middle ear.

In a reversal of application the discrimination score, when relatively high, may be of assistance in the selection of cases of profound mixed deafness as in the case of the patient whose audiogram is shown in Figure 2. This 45-year-old woman gave a history of bilateral rapidly progressive deafness for the past 27 years. As the audiogram in Figure 2 shows, her loss of hearing was severe bilaterally, with markedly depressed bone conduction. Her discrimination scores were taken at levels of 10 and 15 db. above the speech reception thresholds of the right and left ears respectively, which were 110 db. on the audiometer, the limit of the equipment. In view of the profound hearing loss, the resultant scores of 58% and 62% suggested good cochlear reserve, and an operation was performed. Again, the main criterion for selection was the substantial air-bone gap. Following mobilization of the rigid left stapes, her average pure-tone loss in that ear rose from 83 to 68 db. and her speech reception threshold from 95 to 75 db. The discrimination score on the ear that had been operated on was tested postoperatively at the same audiometric level as in the preoperative test, 110 db. on the audiometer. This was now 35 db. above her new speech reception threshold. The dis-

Fig. 6.—Gellé apparatus designed by Thullen and used by the authors to determine stapes fixation, is shown coupled with the Peters SPD2 audiometer.

crimination score was 94%, seeming to justify the preoperative assumption of good cochlear reserve. The modest improvement in threshold made a considerable difference in her ability to profit from the use of a hearing aid.

Testing for Stapes Fixation

When the present era of stapes surgery began with Rosen's [3] report in 1952, followed by subsequent reports by Rosen and others on mobilization at the stapedial neck and at the stapedial footplate, there seemed to be a need for determining with some degree of certainty not only whether the stapes was in fact ankylosed but also the degree of fixation.[4] Several methods for obtaining such information have been considered by us. The first of these was the classic Gellé test modified to give quantifiable readings. Figures 6 and 7 show the equipment (devised by Thullen) as observed by us in Zöllner's clinic in Freiburg.

The equipment was designed to provide a measurable amount of air pressure trapped against the tympanic membrane. A tone is presented through the same tube from an air-conduction earphone which forms one end of the air cylinder and which is activated by a Peters SPD2 audiometer modified to show hearing loss in 1 db. steps. The tube is held firmly in the external canal by an inflatable rubber cuff over the main tubing. This permits relatively high pressures to be used against the drum membrane. As in the classic Gellé, two measures were made

Fig. 7.—The Gellé apparatus is shown in use with a patient.

with the test tone, one with no air pressure on the drum, the other with pressure. Thresholds for 1000 cps were obtained under both conditions. The two conditions were then employed again, this time for measurements by bone conduction. Since the purpose of the measurements was to note threshold shifts rather than absolute values, the calibration of the air and bone systems under the conditions of use was not attempted.

While our investigations with this quantifiable Gellé test were under way, Rosen devised the fenestra ovalis technique for use when a rigidly fixed stapes defied attempts at indirect (stapedial neck) or direct (stapedial footplate) mobilization.[5] It seemed irrelevant, therefore, to relate surgical successes and failures to the Gellé findings. With the new method of fenestrating the oval window through a solidly fixed stapedial footplate or where the crura had been fractured, it became apparent that the degree of fixation no longer mattered as much. It seemed that the only remaining function of a test for stapes mobility was to rule out surgery in cases in which the stapes was mobile. Since in more than 2000 cases selected for stapes surgery over the past five years by the air-bone gap criterion, a freely mobile stapes has been encountered in only five patients, the applicability of this information seems minute.

It is probably true that the less rigidly fixed footplate has a better chance for mobilization at the neck or at the footplate, but if there is any degree of stapes ankylosis, surgery seems indicated. Now, even in cases where the footplate is extremely rigid, a fenestra ovalis can be made through the footplate and the hearing restored. For example, in one case (Fig. 5, previously shown) the Gellé test showed maximum shift by our standards, indicating a mobile stapes. Nevertheless, at surgery palpation revealed a moderately fixed stapes. In this case, mobilization at the footplate brought a 30 db. improvement in hearing, as shown in Figure 5.

At the present state of our development in the use of this Gellé apparatus, we do not feel that we can rely upon the test findings for a clear contraindication of surgery. The investigations are being continued, however, by J. Guerriero, our audiologist.

Prediction of Results

The prediction of results following fenestration of the lateral semicircular canal has often been based upon the acceptance of a residual hearing loss due to the elimination of the intact ossicular chain and other

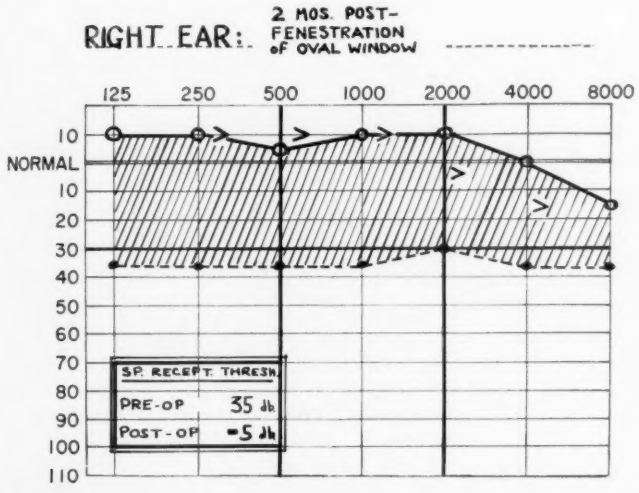

Fig. 8.—In this case the crura were found to be extraordinarily strong and resisted pressure without fracturing. The incus was removed to permit a better view of the footplate, which was then fenestrated. After fenestration of the oval window, the stapes remained as rigid as before, but despite this rigidity the patient's hearing was returned to the bone level, in this case resulting in better than average hearing.

mechanical factors. This does not seem to obtain in the case of mobilization of the stapes either at the stapedial neck or at the footplate. Furthermore, experience with the fenestration of the oval window indicates that such a fenestra can result in closure of the air-bone gap, as in the case shown in Figure 8.

It appears that any of the three techniques developed by Rosen—mobilization at the neck, mobilization at the footplate, and fenestration of the oval window through the footplate—can result in closure of the air-bone gap. Guillon of Paris also reported similar results at the Sixth International Congress of Otolaryngology in Washington, D. C., in May, 1957.

Summary of Conclusions

The ultimate goal of treatment of bilateral otosclerotic deafness is the restoration of hearing in both ears. In view of the effectiveness of the various operations upon the stapes in increasing the number of successes, it is suggested that the patient be advised to look upon stapedial surgery as a course of treatment to be continued and when necessary repeated, until the maximum bilateral restoration of hearing has been accomplished.

At the present time, the most useful tests for a determination of suitability for stapes surgery appear to be the pure-tone air- and bone-conduction thresholds. It is hoped that new tests will be developed for further refinement in the selection of cases and the prediction of results.

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A Study of Hearing Acuity in Adult Aphasic and Cerebral Palsied Subjects

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Introduction

The problem of aphasia, which was first described by Broca [32] in 1870, has been studied extensively during the intervening years. In particular, disturbances in auditory comprehension, with reference to receptive aphasia, have been minutely investigated. Likewise, a vast amount of literature is available pertaining to the expressive and mixed forms of the disorder.[16,17,21]

Aphasia has been defined as a symbolic language impairment resulting from an organic disturbance of cortical tissue.[32] Since the overt manifestations of the cortical insult are primarily observable as symbolic disruptions in either expressive or receptive language patterns, or both, the question arises as to whether or not a concomitant reduction in auditory sensitivity may accompany the condition.

Another form of cerebral injury, not specifically associated with symbolic language disturbance, is encountered in cerebral palsy. Cerebral palsy may be defined as a neuromuscular and sensory handicap, caused by damaged or absent brain structures.[11] It is usually classified on the basis of the following five categories, namely, spastic, athetoid, and ataxic palsy, rigidity, and tremor. The highest incident of hearing loss is reported to occur in the athetoid classification,[18] although the spastics as a group have more hearing defects than persons in the general population.[21]

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Background of Study

A review of the literature on adult aphasia reveals only one specific attempt, and that by Street,[29] to study hearing acuity. Street administered pure-tone audiometric tests through the frequency range 125-12,000 cps to 90 aphasic patients ranging in age from 19 to 70 years. The etiology included cerebrovascular accidents, traumatic injuries, and tumors. All of the subjects were reported to have some degree of comprehension difficulty. The results showed that 80 of the 90 subjects had hearing losses, 44 having reduced acuity through the speech frequencies.

In order to get an indication of the effects of age upon the observed number with hearing losses, Street grouped her subjects according to whether they were less than or more than 50 years of age. She found that 37 subjects with either speech-frequency or high-tone hearing loss were less than 50 years old as compared with 43 subjects in the older age category. The large number of subjects with hypacusis in the younger age group suggests that hearing loss might be associated with aphasia. Although Street apparently had no premorbid audiometric data, nevertheless case histories of her subjects which were obtained either from the patients themselves or members of their families frequently revealed no recognizable hearing loss prior to the accident.

Next, Street grouped her subjects on the basis of etiology. She found that 51 of the cerebrovascular group, 17 of the traumatic group, and 12 of the tumor cases had either high-tone or speech-frequency losses. Such findings as Street's suggest that hearing loss is a concomitant of aphasia-producing lesions.

Although Dandy [12] did not specifically refer to aphasia and hearing loss, nevertheless he postulated that a lesion, either central or cortical, could not have an influence on hearing acuity. He goes on to say that removal of either the right or left temporal lobe has no effect on audiometric hearing tests. In addition, removal of the entire right cerebral hemisphere (this has been done seven or eight times [12]) has not the slightest effect on hearing.

Animal experimentation offers support to Dandy's contention.[7] It has been shown, for example, that the removal of either the right or the left cerebral cortex of dogs induces only a 2 to 5 db. reduction in hearing acuity. When however, the entire cortex is ablated, the ensuing loss is of the magnitude of 70 to 75 db.

Drury [13] studied hearing acuity in subjects with brain tumors. None of this group manifested symptoms of aphasia. He reported reduced acuity for pure tones, particularly in the high frequencies. Conversely, Bocca et al.[6] found normal hearing in their cases of cerebral tumor.

Winchester and Hartman [33] investigated auditory dedifferentiation in dysphasic subjects. Dedifferentiation, as defined by Goldstein,[17] is a breakdown in the ability to distinguish between the foreground and the background of a given sensory, motor, or ideational configuration. Winchester and Hartman found that brain-injured subjects repeated word lists as well in quiet as their non-brain-injured controls. When however, background noise was introduced, the dysphasic subjects were definitely inferior, manifesting a breakdown in auditory dedifferentiation.

The conflicting findings with reference to hearing acuity in brain-damaged subjects, as well as the relative paucity of studies pertaining to the various etiological classifications, points up the need for continued research in these areas.

Most of the literature which deals with the hearing acuity of cerebral palsied subjects pertains to children. In general, there is unaminity in the results. Since the condition usually does not vary with age, the hearing acuity of adult cerebral palsied patients, except as altered by acoustic trauma, presbycusis, or some other disease of the ear, would be expected to be similar to the hearing acuity of children of like classification.

Rutherford [23] tested 168 cerebral palsied children and found hearing loss in 41%. Neither the frequencies at which the testing was done nor the types of losses were reported. She stated that the reactions of the cerebral palsied children were such as tended to indicate that not all of the hearing problems were due to hearing loss per se, but a part of the larger picture of cerebral palsy.

In comparing the hearing losses of spastics and athetoids, Saltzman [24] noted that the former tended to have slight losses which were more marked in the lower frequencies.

Asher [3] found hearing loss in 22 out of 24 athetoid children with histories of neonatal jaundice. Conversely, only 4 out of 18 athetoids without neonatal jaundice showed hearing loss. In a group of spastic children, only two had hearing losses severe enough to delay speech, none of which was of the high-tone type characteristic of his athetoid group.

In studying RH deaf athetoids Hardy [18] found the higher frequencies to be much poorer than the low tones. The hearing losses were of the nerve or perceptive type.

In view of the research to date, the present investigation was concerned, first, with a comparison of the hearing acuity of aphasic and cerebral palsied adults; second, with a comparison of each of the aforementioned groups with a control group of normally

hearing subjects; third, with a comparison of the left and right ears of each group, and fourth, with a comparison of the left and right ears of the aphasic group when the subjects were classified on the basis of dominant and nondominant hemisphere lesions.

Since the cause of the aphasia in 34 out of a total of 37 cases was cerebrovascular insult, and since a diagnosis of spasticity had been made in 20 of the 26 cerebral palsied subjects the comparison between the brain-damaged groups was essentially one of etiology. The specific questions for study were then as follows:

1. Are there statistically significant differences between the auditory pure-tone threshold of normally hearing adults and aphasic adults of cerebrovascular etiology? Between normally hearing adults and cerebral palsied adults of the spastic type?
2. Are there statistically significant differences between the auditory pure-tone threshold of adult aphasics and adult cerebral palsied subjects of the previously referred to etiologies?
3. Are there statistically reliable differences between the ears of each experimental group?
4. What is the effect upon the auditory sensitivity of the two ears of a lesion in either the dominant or the nondominant hemisphere of aphasic subjects with cerebrovascular etiology?

The Subjects

Seventy-three adult subjects were involved in this study. Of this number 37 had been diagnosed as aphasic, and 26 as cerebral palsied. The remaining 10 subjects were nonbrain injured adults with normal hearing who served as a control. The mean age of the aphasic group was 52.8 years, with a range of 33 to 86 years. The mean age of the cerebral palsied subjects was 25.2 years, with a range of from 16 to 38 years; and for the normals, the mean age was 31.2 years, with a range of 22 to 42 years.

The precipitating causes of the aphasia symptoms were cerebrovascular accident in 34 cases, traumatic injury in one case, and brain tumor in the remaining two cases. Since the audiometric patterns of the brain-injured and tumor cases were not different, the total number of cases were used in the statistical treatment of the data except as indicated. Of the 34 cases who had suffered cerebrovascular accident 24 were right hemiparetics. The remaining 10 cases were left hemiparetics. All, however, were right-handed prior to their illness. In so far as handedness is an index of cerebral dominance, 24 of the subjects could be regarded as having lesions of the dominant hemisphere and 10 as having lesions of the nondominant hemisphere. All of the subjects were receptively as well as expressively involved. They had been classified, however, by the Chief Neurologist of the Veterans' Administration Hospital in Kansas City, Mo., and by the Physiatrist at the Rehabilitation Institute of Kansas City, Mo., as predominantly expressive aphasics.

Case histories of the aphasic subjects revealed that none had been aware of a hearing loss prior to the cerebral accident. In addition, medical examinations of the ears were negative. Consultations with the families of the subjects failed to bring to light any awareness of a previous hearing loss. Three of the subjects had been exposed to noise in the armed forces, but none exhibited indications of acoustic trauma in their audiograms. In the absence of preinjury audiometric data it seems justifiable to assume that the subjects' hearing acuity for the speech frequencies 500-2000 cps was within normal limits before their accident.

In the cerebral palsied group there were 20 spastics, 3 athetoids, 2 ataxics and one with tremor type palsy. Two of the subjects reported that they had had middle ear infection in childhood, which, however, had been successfully treated. The remainder had experienced no ear disease in so far as they could remember. Only one subject of the total number of aphasic and cerebral palsied subjects (63 subjects) had a severe enough hearing loss through the speech range to justify the use of a hearing aid.

The aphasic subjects had been referred for this study by the Kansas City, Missouri, Veterans' Administration and the Rehabilitation Institute of Kansas City, Mo. The cerebral palsy subjects were referred by the Challenging Pioneers Club, a social organization of cerebral palsied adults, and the Goodwill Industries. All of the cerebral palsied subjects were gainfully employed in occupations suggesting normal or near-normal intelligence.

Audiometer, Calibration, Test Chamber, and Procedure

The instrument used for the tests was a commercially available Maico F-1 audiometer which had been factory-calibrated to American Standards Association specifications for sound-pressure output, frequency, and linearity of the attenuator[19] immediately preceding the study. Constancy of the output and the linearity of the attenuator were checked periodically during the collection of the data by means of an Allison Artificial Ear Assembly at the Kansas University Medical Center

Hearing Clinic. The audiometer was equipped with a head band and a pair of matched ANBH-H-1A earphones, mounted in MX-41/AR cushions.

The tests were conducted in a quiet room at the Missouri Rehabilitation Institute. This room had an ambient noise level of 38 db. on the A scale of a General Radio Sound Level Meter, Type 1551-A. Unfortunately, an octave-band analyzer was not available to specify in octave bands the level of the ambient noise. Repeated tests on subjects with normal hearing in this room, as well as the mean threshold readings for the control group of normally hearing subjects in this study, suggest that the peak energy in the ambient noise lies in the frequencies below 250 cps. That is to say, normal thresholds are consistently obtained for all frequencies by air conduction with the exception of 125 cps. At this frequency, clinicians who use the room daily for hearing tests report a shift of about 10 db. from the normal zero reference. The mean 9 db. loss at 125 cps for the control group in the present study tends to confirm the observations of the clinicians. Since MX-41/AR cushions afford about a 20 db. attenuation through the speech range, and as much as 35 db. at the higher frequencies, the influence of ambient noise, except as discussed, is regarded as minimal.

Thresholds were determined by the clinical ascending method at frequencies 125, 250, 500, 1000, 2000, 4000, and 8000 cps. The customary order of frequency presentation was observed, namely, 1000, 2000, etc.

In the analysis of the data nonparametric statistics were employed[27,30] because Hartley's test of variance suggested the presence of heterogeneity of variance.

Results

In the Table are shown the mean thresholds and standard deviations in decibels for the right and left ears of the normal, aphasic, and cerebral palsied subjects. The approximately 9 db. loss at 125 cps of the control group has been mentioned previously, and is attributable to the low frequency ambient noise of the test chamber. With the exception of 8000 cps in the right ear, the mean thresholds of the control group were reasonably close to the National Bureau of Standards reference levels for average normal hearing.

In the case of the aphasic subjects the magnitude of the deviations from the norms at each frequency is comparatively large for each ear. The pattern of the loss for the left and right ears is remarkably similar. It will also be noted that the standard deviations for the frequencies 2000-8000 cps are exceptionally large, evidencing great variability through the higher range.

Means and Standard Deviations in Decibels for the Right and Left Ears of the Control Group of Normal Hearing Subjects and of the Experimental Groups of Aphasic and Cerebral Palsied Subjects

Freq.	Normals N 10		Aphasics N 37		Cerebral Palsy N 26	
	Mean	S. D.	Mean	S. D.	Mean	S. D.
			Right Ear			
125	8.50	3.37	19.32	9.66	18.83	11.77
250	−0.50	4.97	12.70	9.47	11.35	8.43
500	0.00	6.67	12.84	12.56	11.73	7.69
1000	1.50	4.74	12.16	11.82	9.81	6.24
2000	−2.00	6.32	11.89	15.56	3.85	8.40
4000	2.00	9.19	27.57	17.97	6.25	10.06
8000	6.50	9.14	30.68	22.43	10.90	15.43
			Left Ear			
125	9.50	4.38	20.00	8.74	16.35	8.31
250	0.00	5.77	12.16	8.30	10.77	9.24
500	−1.00	5.68	10.81	8.36	10.78	7.57
1000	−1.00	4.59	11.35	9.18	7.31	9.41
2000	−1.00	5.68	13.38	15.14	4.81	9.85
4000	3.50	9.73	31.08	20.65	7.50	14.94
8000	2.00	7.89	33.92	24.18	10.38	17.66

Note: The reference level at each frequency is the NBS sound pressure for average normal threshold.

The mean thresholds and standard deviations in decibels for the cerebral palsied subjects will be seen in the last two columns of the table. Again, there is a pronounced deviation at most frequencies from the control group. In general, the deviations are not as large as in the case of the aphasic group.

To point up the differences between the groups Figure 1 was prepared. Cursory inspection reveals immediately that the cerebral palsied group had less hearing loss in the high frequencies than the aphasic subjects. One also notes the pronounced low-frequency loss of the cerebral palsied subjects and the marked stiffness-like tilt of the curves for both ears. It will be recalled that the majority of subjects in this group were of the spastic type. These results would tend to support the observations of Saltzman,[24] that spastics appear to exhibit low frequency losses.

In order to determine whether or not the differences in threshold sensitivity between the control group of normal hearing subjects

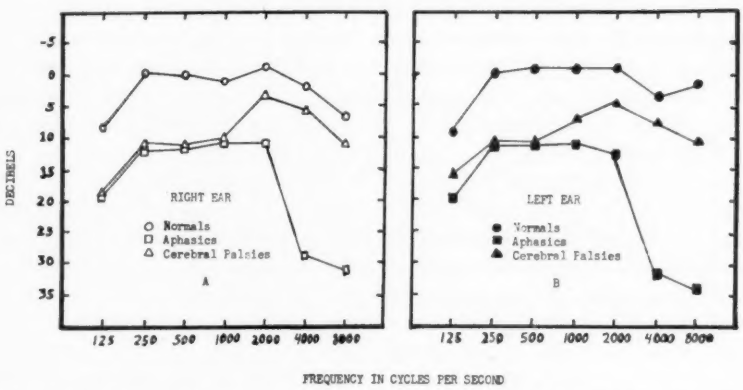

Fig. 1.—Mean thresholds for normal, aphasic, and cerebral palsied subjects for the right and left ears with reference to the normal average threshold.

and the two experimental groups were statistically significant, z scores were computed.[30] The z scores for the differences between the means of the normals and of aphasics were significant at the 1% level of confidence for both the left and the right ears at all test frequencies. In other words, there is very strong statistical evidence that a real difference in threshold acuity exists between the two groups for all frequencies at which measurements were made.

Next, the cerebral palsied subjects were compared with the normal group. The differences between the means for both the right and the left ears were significant at the 1% level of confidence for frequencies 125, 250, 500, and 1000 cps. Conversely, no statistically significant differences were found for frequencies 2000, 4000, and 8000 cps in either ear. These results offer statistical confirmation of low-frequency loss in cerebral palsied subjects of the spastic type.

In the comparisons between the two experimental groups the z scores of the differences between the mean thresholds reached significance at the 1% level only at frequencies 4000 and 8000 cps. The results were similar for the both ears. Another significant finding was at 2000 cps in the left ear, but the level of significance barely reached 5%. The cerebral palsied and aphasic subjects were therefore alike in threshold sensitivity through the frequency range 125-2000 cps.

The left and right ears of each group were next compared, with none of the groups exhibiting statistically significant differences between ears at any of the test frequencies.

Finally, the 34 aphasic subjects who had suffered cerebrovascular insult were grouped on the basis of having either a right or a left hemiparesis. Twenty-four of the subjects fell into the former classification and the remaining ten into the latter. All of the subjects had been right-handed prior to their accident. On the assumption that handedness is indicative of cerebral dominance, 24 subjects could be considered as having suffered insult to the dominant hemisphere, with 10 possessing nondominant hemisphere injury. The mean threshold differences at the test frequencies between the left and the right ears of each group were therefore treated statistically to determine whether or not the factor of cerebral dominance influenced threshold sensitivity. In short, none of the z scores for the differences between the ears of either group was significant at the 1% level of confidence. Within the limits of this study the results indicate that there is no difference in the auditory threshold between ears, regardless of whether the lesion is in the dominant or the nondominant hemisphere in subjects with left hemisphere

cerebral dominance. Furthermore, when the differences between the mean thresholds for the two groups were analyzed statistically, none was significant at the 1% level of confidence. Hence, both groups evinced similar hearing-loss curves.

Decreased hearing acuity as a result of advancing age appears to be a well-established fact.[8,28,31] Recently, the Subcommittee on "The Relations of Hearing Loss to Noise Exposure" [1] has published curves for men and for women showing the average loss in hearing acuity which is expected with increasing age. Since presbycusis, or hearing loss due to advancing age, does not occur in every person of advanced years, curves of hearing sensitivity relying upon an average correction factor would have to be applied with caution. For example, approximately half of the aphasic subjects did not manifest the degree of hearing loss, particularly in the high frequencies, which would be expected as a result of their age. Rigid application, therefore, of an average correction factor for presbycusis based on age, regardless of a subject's actual acuity, would have produced somewhat ludicrous results in many instances. In other words, after correction for presbycusis many of the aphasic subjects would have had sensitivity curves which were superior in the higher frequencies to the Sivian and White thresholds for best normal hearing. While it is not improbable that some of the subjects had very superior hearing in their youth, nevertheless it seemed to us more reasonable to make corrections for presbycusis only when there was strong suggestion from the audiogram that presbycusis was present.

Since the age range of the cerebral palsied subjects was below the minimum age level at which changes in auditory sensitivity on the average are noted, corrections for presbycusis were not applied. With reference to the control group of normals, the only corrections required were at 4000 cps. Conversely, the aphasics required corrections at frequencies 500, 1000, 2000, and 4000 cps. Corrections at 8000 cps were not attempted since none was available for male and female subjects separately. Bunch's data at 8000 cps combined the sexes, whereas the other two large hearing surveys, the World's Fair [28] and the San Diego County Fair,[31] collected data at 7040 cps.

The mean threshold curves after correction for presbycusis as discussed above are shown in Figure 2. The general similarity of the aphasic and cerebral palsy curves is at once apparent. Although statistical analysis of the differences between the means of the two experimental groups could not now be appropriately applied because of the nature of the corrections, nevertheless it is of interest to observe the emergence of the stiffness-like tilt in the aphasic group. To

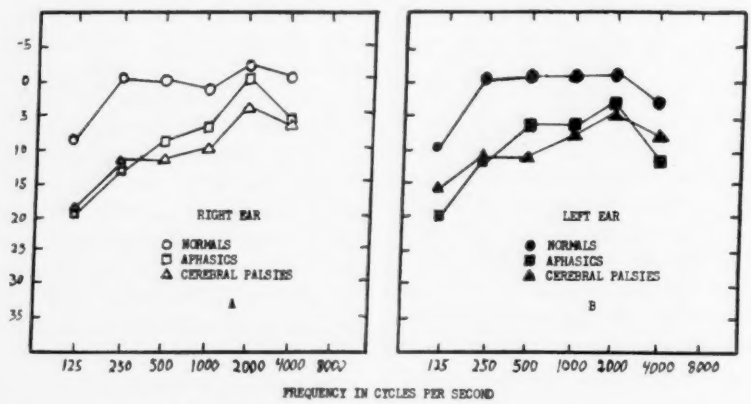

Fig. 2.—Mean thresholds for normal, aphasic, and cerebral palsied subjects for the right and left ears after corrections for presbycusis.

reiterate, the two brain-damaged groups of different etiology evince similar hearing sensitivity curves after correction for presbycusis. As was stated, no corrections for presbycusis were made at 8000 cps. However, it is clear by referring to Figure 1 that had corrections been employed of the magnitude provided for 4000 cps, a corrected curve at 8000 cps for the aphasic group would not have been poorer than about 20 db.

Another point which bears mentioning is that neither experimental group has an average hearing loss in excess of 10 db. in either ear for the frequencies 500 to 4000 cps after the correction. The largest uncorrected average hearing loss for the frequencies 500-2000 cps inclusive was only 12.29 db., which figure is usually regarded as being within the limits of normalcy for the range. With regard to the upper frequencies (4000-8000 cps), the average uncorrected hearing loss would usually be considered as only of slight to moderate degree.

Comment

Bone-conduction measurements were not made in this study. However, the otological examinations of both experimental groups were negative. Furthermore, only two of the experimental subjects had experienced middle ear infections. In both instances the infections had occurred in childhood and had been treated successfully without subsequent awareness of hearing loss. It seems reasonable, therefore, to assume that the depression in acuity in the low frequencies of the aphasic and cerebral palsied subjects is not the aftermath of a conductive component.

In the absence of premorbid audiometric data as in the case of the aphasics, generalizations contingent upon threshold curves which were corrected for presbycusis are, of course, tenuous. One might speculate, however, that cerebral insults such as studied here, although producing manifestly different behavioral symptoms, nevertheless induce identical effects on hearing acuity. The nonsignificant differences between the thresholds of the dominant and nondominant hemisphere lesion groups (the aphasics) as well as the nonsignificant differences between the left and the right ears of each group, plus the nonsignificant differences between the aphasics and the cerebral palsied for the frequency range 125-2000 cps, might suggest a generalized effect of the particular types of brain injury (cerebrovascular and spastic), regardless of locus, on hearing acuity. In other words, the losses in the lower frequency range as observed may be related to poorer ability to attend and concentrate—possibly a concomitant of the types of brain injury.

It will be recalled that Winchester and Hartman [33] found dysphasic subjects performed more poorly in noise than in quiet in their responses to word lists. These authors attributed the discrepancy to a breakdown in auditory dedifferentiation as a result of the noise. As was pointed out earlier, the test chamber used in the present study appeared to have an ambient noise level sufficiently high to induce about a 10 db. shift in threshold at 125 cps for the control group of normal hearing subjects. The pronounced losses in the low frequencies of both experimental groups (in particular at 125 cps) possibly suggest poor ability to dedifferentiate the signal in ambient noise.

Summary

With reference to the questions which were posed relative to the study, the findings are as follows:

1. Differences in hearing acuity for pure tones by air conduction between the control group of subjects with normal hearing and the experimental group of aphasic subjects were significant at the 1% level of confidence for all frequencies and for both the left and the right ears.

Differences in hearing acuity for pure tones by air conduction between the control group of subjects with normal hearing and the experimental group of cerebral palsied subjects were significant at the 1% level of confidence for frequencies 125, 250, 500,

and 1000 cps in both the left and the right ears. Differences at 2000, 4000, and 8000 cps were nonsignificant. These results provide evidence for Saltzman's observations that cerebral palsied subjects of the spastic type tend to have low-frequency losses.

2. Differences in hearing acuity as described between the aphasic and cerebral palsied subjects were significant at the 1% level of confidence for both ears only at frequencies 4000 and 8000 cps.

3. There were no statistically significant differences between the left and the right ears of any of the groups.

4. There were no statistically significant differences between the left and the right ears of aphasics with cerebrovascular etiology, regardless of whether the lesion was in the left dominant or right nondominant cerebral hemisphere as designated in this study. Further, there were no significant differences in threshold acuity between the dominant hemisphere lesion and nondominant hemisphere lesion groups.

5. After correction for presbycusis the hearing loss of the aphasic subjects was similar to that of the cerebral palsied group. The average hearing loss through the speech frequencies 500-2000 cps inclusive was less than 15 db. before as well as after correction for presbycusis. Decreased ability to attend and concentrate, a possible concomitant of the particular types of brain injury which were studied, and decreased ability to dedifferentiate the foreground signal in background ambient noise, were suggested as possible reasons for the observed hearing loss in both experimental groups.

Rainbow and 39th Sts.

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Case Reports

Synovial Otorrhea

NED I. CHALAT, M.D., Detroit

I would like to report an unusual fracture of the temporal bone complicated by persistent otorrhea and to discuss the importance of accurate identification of the discharging fluid in such an injury. The patient was encountered during a tour of duty as otolaryngologist with the U. S. Air Force and that service provided me with the facilities to manage the case and record the data.

The patient, a 29-year-old U. S. A. F. pilot, was seriously injured at 2:10 a. m. on July 25, 1953, while riding as guest passenger in an automobile driven by a friend. He was taken immediately to a neighboring U. S. Army Hospital, where he was treated for a compound fracture of the skull (petrosa) with a complete left facial nerve paralysis. He also had lacerations about the face, tongue, and cheek, and there was a (cerebrospinal fluid) otorrhea. Five days later, a tarsorraphy was performed on the left eye, and after two weeks he was transferred to our service at Parks Air Force Base Hospital.

The patient was extremely apprehensive when first seen. There was a well-healed left tarsorraphy, complete left peripheral facial paralysis, and several healed lacerations about the left side of the face, one parallel to the ramus of the mandible. A purulent discharge was present in the left external auditory canal. There was no meningismus.

Our first consideration was the purulent otorrhea. Smear, culture, and sensitivity studies revealed a Gram-negative bacillus and a Staphylococcus, both of which were sensitive to penicillin and chloramphenicol (Chloromycetin.) On subculture, however, the Gram-negative bacillus was lost. The Staphylococcus was identified as aureus, hemolytic type.

Penicillin and chloramphenicol were started, and the discharge from the left ear soon became odorless and colorless, but did not abate. The extensive collapse and maceration of the external canal prevented examination of the tympanic membrane.

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While the patient was on this regimen physiotherapy with stimulation of the muscles of the face was initiated. Faradic and galvanic testing revealed a reaction of degeneration. This was later verified by electromyelography.

Two months after the injury, the facial paralysis was still present, and the clear, colorless otorrhea persisted. Repeated cultures of the fluid showed no growth. Surgical intervention was decided upon, and a Y incision was made postauricularly and along the anterior border of the left sternomastoid muscle. A cortical mastoidectomy was done, and complete careful examination of the entire dural plate failed to reveal either a fracture or the source of the "cerebrospinal otorrhea." The facial nerve was then identified in the middle ear, and a decompression begun. In the third portion of the nerve, in the temporal bone, as it approached the stylomastoid foramen, an organizing hematoma was found completely circumscribing the nerve. Above this area, the facial nerve, quite edematous, was readily identified. Distally, towards the stylomastoid foramen following incision of the periosteum, no definite fibers could be found even with magnification. The hematoma was carefully dissected from about the nerve, and upon its removal a fracture line was first seen on the facial ridge. This extended anteriorly along the inferior margin of the external auditory canal and forward directly into the temporomandibular joint. Synovial fluid was now seen to ooze through the fracture line. A bony displacement in the external auditory canal was repaired, and the denuded area over the fracture near the temporomandibular joint was covered by skin graft transferred from the margin of the incision. An attempt was made to follow the nerve to and beyond the foramen but was abandoned because the remnants were too minute and the risk of injury too great. A drain was inserted and the wound was closed.

With the cause of the difficulty disclosed and removed, the patient made an uncomplicated postoperative recovery. He was given daily massage and stimulation of the facial muscles. Almost exactly five months after the operation, the first voluntary movements and most of the involuntary movements of the face returned. He was returned to duty eight and a half months after

the accident, but was not immediately returned to flying status because of persistent lack of involuntary control of the left eyelid.

Comment

A search of the literature failed to disclose instances of synovial fluid otorrhea following trauma probably because the clear, colorless discharge was generally reported as cerebrospinal fluid. In view of the experience with this case, more accurate identification of the fluid is indicated in similar cases. Cerebrospinal fluid drainage would be augmented by increasing the intracranial pressure even with the external auditory canal collapsed. Laboratory studies of the fluid for its sugar and protein content would further promote its identification.

The fracture here evidently occurred along with the longitudinal group of injuries described. It paralleled the long axis of the petrosa, did not approach the area of the geniculate ganglion, and involved the tympanic portion of the temporal bone almost exclusively, extending from the facial canal to the tympanic fissure.

Summary

A case is reported of a patient who presented an unusual fracture of the temporal bone complicated by facial paralysis and a persistent otorrhea with a discharge of clear fluid. At operation a hematoma surrounding the third portion of the facial nerve was removed, and a longitudinal fracture extending from the facial ridge into the temporomandibular joint was uncovered. Synovial, not cerebrospinal fluid, oozed from the fracture line. This might have been revealed had the fluid been more carefully studied. Complete recovery followed surgical treatment.

1553 Woodward Ave.

Foreign Body in the Larynx

SIDNEY SPECTOR, M.D., and ARISTON BAUTISTA, M.D., New York

From time immemorial doctors have been called upon to deal with children who have either swallowed or aspirated foreign bodies commonly found in their environment. The public is generally aware of the extraordinary tendency that little ones have for placing things in their mouths and, as a result, such children are usually brought to the doctor with the history that a coin, button, pin, etc., had disappeared from sight and the parent is sure it had been either swallowed or aspirated. The physician, with a few simple questions, observation of symptoms and examination, sometimes including x-ray, usually quickly confirms the diagnosis and institutes immediate treatment.

There are cases, however, where the parent is unaware of the possibility of an aspirated foreign object, and in such cases the child is brought in with only a statement of symptoms of comparatively sudden onset. Recently, I encountered a case where a 9-month-old child developed mild respiratory symptoms, and because a foreign body had not been suspected, the diagnosis was delayed for 10 days.

It seems to me that such a case is worth reporting because the complications of an unrecognized foreign body in either upper air or food passages may be so severe as to threaten life, while a recognized foreign body can, in the vast majority of instances, be comparatively simply removed by proper endoscopic techniques.

Figure 1

A 9-month-old white boy was admitted to the Kingston Avenue Hospital for Infectious Diseases on May 14, 1956, with a history of cough, hoarseness, noisy respirations, and fever of 10 days' duration. The father stated that on about May 4, 1956, he was playing with the child in the crib when the child suddenly had a coughing spasm which subsided within about two to three minutes. At the time, the parents thought the baby was developing a "cold" and attributed the sudden onset of cough to mucus in the throat. For the rest of the day and night, the child had an intermittent dry cough, noisy respirations, and an intermittent hoarse cry. He was taken to the outpatient department of a nearby hospital where a diagnosis of an upper respiratory infection was made, and the child was given nose drops and a cough medicine. For the following eight to nine days, these symptoms persisted, sometimes becoming severe, but most of the time of moderate degree. A temperature of 100 F to 101 F was present daily. The child was brought back to the same clinic on two more occasions, and the same treatment was given.

On the day of admission to the Kingston Avenue Hospital, the child was taken to a private physician because of the persistence of the symptoms, and he recommended that the child be admitted to a

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Assistant Professor of Otolaryngology (Dr. Spector) and Instructor in Otolaryngology (Dr. Bautista), State University of New York Medical Center at New York City.

Figure 2

hospital with a diagnosis of "croup." On admission, the temperature was 102 F, the pulse was 100 and respirations were 36 per minute. The color was good. There was a mild stridor, both inspiratory and expiratory, and mild suprasternal and epigastric retractions were present. The cry was hoarse, and a mild intermittent cough was present. The nutrition of the child was good.

Routine examination of the nose and throat were essentially negative. The child was placed in a croup tent, and x-rays of the neck and chest were taken (Figs. 1 and 2). The x-ray report read as follows: There is an open safety pin in the upper trachea with the open end pointing cephalad. The point of the open pin appears to be imbedded in the tracheal wall. The pin is in the upper trachea overlying C-2 and C-3. The lungs are clear.

A direct laryngoscopy was immediately done, and, on direct vision, the pin was noted to be open and lying within the glottic chink. The pocket of the pin was proximal and was lying on the left vocal cord while the open point was imbedded in the inferior surface of the right vocal cord. The angle of the pin was lying within the tracheal lumen. A laryngeal alligator forceps was used to remove the pin by grasping the pocket end and maneuvering the point out of the right vocal cord.

After the procedure, the stridor and hoarseness immediately disappeared. Despite the presence of the pin for 10 days, there was remarkably little edema of the larynx. The postoperative course was afebrile and uneventful, and the child was discharged from the hospital on May 19, 1956.

This case history demonstrates the need for us to remember the possibility of foreign bodies in the upper air and food passages in children who develop symptoms referable to these regions. If thought of, the diagnosis is usually easy, and effective therapy can be quickly given with an excellent chance for rapid and complete cure.

225 Eastern Parkway, Brooklyn.

A New Gag Support for Dissection Tonsillectomy

D. VERNON THOMAS, M.B., Rochester, N. Y.

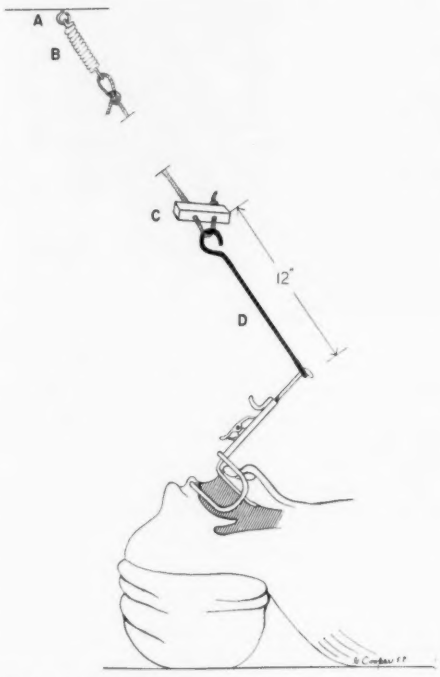
When dissection tonsillectomy is performed with the patient in the supine position under general anesthesia with use of a Davis or Tidings type gag, it is customary for the anesthetist to hold the handle of the gag. Unless this is maintained in the optimum position, both the patient's airway and the surgeon's view of the operative field are compromised. Yet holding the gag in this fashion is tiring, and it is not unusual for an anesthetist to exhibit a fatigue-tremor in the hand and forearm after doing several cases on the same day.

In my view, the demands of this purely mechanical task detract from the anesthetist's ability to pay adequate attention to his patient's general condition; when the anesthetist does make adjustments to the anesthesia machine, take blood pressure readings, keep a record, etc., he is likely to move the gag and hinder the surgeon's work.

I have tested several devices intended to support the gag handle, including those described by Draffin[1] and by Thomson and Negus,[2] but have found none of them to be satisfactory in all patients or for all surgeons. Draffin suggested a "bipod" whose two feet rested on the operating table drapes alongside the patient's head and whose apex was connected to the handle of the gag. In our experience this arrangement proved to be unstable and liable to slip, while a further inconvenience was that two or three sizes were necessary to serve for patients of different ages.

Thomson and Negus described a gag support which consisted of a rod connected to a flat metal plate resting on the patient's sternum, but in small patients this device may exert considerable pressure on the chest and embarrass respiratory excursion.

The arrangement which has been used at the University of Rochester Medical Center since September, 1956, is shown in the accompanying illustration. The necessary traction is supplied by suspending the gag from the operating room ceiling by a strong cord into which a spring has been incorporated. The ceiling hook (*A*) is installed a few feet distant from the patient's head so that the angle of pull is between 30 and 40 degrees from the vertical. An 8 in. spring (*B*) of 0.063 in. music wire wound to a diameter of ¾ in. has been found to be satisfactory. The length of the cord is made adjustable by fitting it with a wooden slide (*C*) of the type used for tent ropes, but, in practice, because of the elasticity imparted by the spring it is rarely necessary to change the cord length from one patient to another. The final component is a length of 3/16 in. stainless steel rod (*D*) having at one end a closed loop, and at the other a partly open loop. The loops are approximately 2 in. in diameter and they are in planes at right angles to each other. The over-all length of this connecting rod is approximately 12 in.



Schematic drawing of gag and support in position.

When the surgeon has placed and adjusted the mouth gag, it is maintained in position by connecting its handle to the suspension cord, using the

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From the Division of Anesthesia, University of Rochester Medical Center.

stainless steel rod. The rod is autoclaved and, except for the loop in contact with the cord, remains sterile, so the surgeon himself can make any further adjustments during the operation.

This device has now been used in more than 1000 tonsillectomies performed by several different surgeons and it has proved to be entirely satisfactory. Its advantages are (1) it is suitable, with little or no adjustment, for adults and children alike; (2) it is not hidden beneath the surgical drapes and enables the surgeon to change the placement of the gag at will; (3) the elasticity contributed by the spring allows changes in the height and tilt of the table without disturbing the gag or its support; (4) its stability not only provides the surgeon with a quieter field but also makes damage to the incisor teeth less likely, and (5) it frees the anesthetist to devote his attention to the patient's general condition.

Summary

A new method of supporting the mouth gag used in dissection tonsillectomies is described. The apparatus is simple to construct and to operate and has been tested successfully in more than 1000 cases.

260 Crittenden Blvd.

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Summaries of the Bibliographic Material Available for 1953

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The volume of literature on the subject of deafness is steadily increasing. Many important papers are appearing in other than otolaryngological journals. Some of these are not detected by the reviewers and others are listed in indices under classifications emphasizing a coexisting subject matter so that pertinent data on deafness is not listed. The selection of publications for summary in this paper is based on whether any new or improved aspects of the anatomy, physiology, pathology, clinical examinations, and therapy as pertaining to deafness, are presented. As far as possible an attempt is made to correlate the subject matter so that it does not overlap the work of other reviewers in closely related subjects.

The subject matter has been arranged in the following manner: otosclerosis, Ménière's disease, acoustic and head trauma, miscellaneous contributions on deafness, reconstructive surgery for middle ear deafness, toxic labyrinthitis, vestibular examinations, and the anatomy and physiology of the ear. Recent interest in corrective surgery for middle ear deafness motivated the plan to start a survey of significant material on the anatomy, physiology, pathology, and surgical concepts of this newly emphasized and important surgery. The review is brought to a close with a brief consideration of important papers on related aspects of the anatomy and physiology of the ear. The size of this analysis prohibits inclusion of comments by the reviewers. It is believed that the readers for the most part are interested primarily in the creativeness of the original authors.

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Otosclerosis

According to Shambaugh,[1] six factors have been found to influence osteogenesis at the fenestra. The first was a stimulating effect of bone particles on osteogenesis. This was minimized with the use of irrigating solutions and the microscope. The second factor is the different osteogenic activity of the different bone layers of the labyrinthine capsule. By wide exposure of the inactive and chondral bone layer, the fenestra is unchondralized. The third requisite for a permanently patent fenestra is that it be covered by a thin flap that adheres closely to the fenestral margins. Skin lying against bone tends to inhibit osteogenesis. The osteogenesis-inhibiting property of intact periosteum and the osteum, is taken advantage of by creating a fenestra with intact endosteum up to its knife-like edge so that this endosteum will unite with the periosteum of the skin flap with healing by first intention of the cut bone surface. A fifth factor which contributes toward the permanence of the fenestra is the lessening tendency of a smooth polished bone surface compared with an uneven rough surface to produce new bone. The fenestra is finished by polishing with a smooth burnishing burr. Finally,

new bone tends to invade adjacent young fibrous tissue. Three causes of fibrosis after fenestration are hemorrhage, infection, and a dead space between the skin flap and the bone. Shaping of the contour of the bone so that the fenestra lies on top of the dome causes the skin flap to hug it tightly like a glove. Minimal bleeding is encountered when local anesthetics are used. Shambaugh also states that the differential between the new window and the round window can be increased after fenestration by placing ointment or other material against the tympanic membrane. A most gratifying 5 or 10 db. can thus be obtained.

With increased appreciation of 25 db. of unrestored conductive loss following fenestration and better realization of the need for an adequate preoperative air-bone gap of 40 to 60 db., the considerable number of fenestration failures attributable to incorrect selection should be greatly decreased (Shambaugh [2]). Postoperative serous labyrinthitis with permanent depression of cochlear function ranks as the most frequent and important cause of failure in fenestration operations at present despite an appreciable reduction in the incidence of this complication with improvements in technique. Although one should not expect fenestration to influence the cochlear nerve degeneration associated with otosclerosis, experience suggests that it may be somewhat delayed by successful fenestration. Statistically, the fenestration operation results in a maintained significant improvement of hearing in about 85% of patients followed for six months to 14 years. The final decision as to the value of this improvement to a particular patient depends upon a number of factors, including the degree of loss before the operation, the success that the patient might have had in using a hearing aid, and the hearing needs of the patient in his occupation.

Hutchinson [3] summarizes the essentials to secure a successful fenestration: 1. Adequate steps must be taken to eliminate all nasopharyngeal sepsis. 2. A normal cochlear nerve mechanism must be present at the time of operation. 3. A free functioning round window membrane. 4. To create a fenestra of adequate size in the vestibular region. 5. Insure that the fenestration shall remain adequately patent. 6. Cover the fenestration with a tympano-meatal flap of suitable nature to retain its flexibility. 7. Granulations filling the cavity. The following procedures may give a reasonable result in certain cases of failure: (1) blocked round window—removal of the posterior lip of the round window niche and clearing the niche of adhesions or the creation of a new round window with a plastic flap cut from the floor of the membranous external auditory canal and rotated on its pedicle; (2) perforated tympanic membrane—repaired by plastic procedures; (3) osteogenic closure of the fenestration—a revision operation and reopening of the operation with replacement of the flap; (4) organized sub-flap hemorrhage—a revision operation and removal of the fibrotic mass with reapplication of the flap.

Ogilvie and Hall [4] report their observations on the pathology of otosclerosis. The material studied was obtained from the horizontal semicircular canal of otosclerotics treated by fenestration (125 cases). Signs of otosclerosis were found in the ampullary region in 13% of these cases. Both diffuse and local forms were found as well as various grades of activity. The diffuse damage involves absorption and deposition of the bone through osteoclastic and osteoblastic activity respectively, and thereby leads in sequence to an abnormally cancellous, mosaic, and relatively normal architecture of the bone. The immature bone is basophilic, excessively cellular and woven, while the mature bone is acidophilic, normally cellular and laminated. The local damage (blue mantle) is regarded as a focal manifestation of the diffuse damage. The diffuse disease is classified as active, quiescent, or healed, whereas the local damage is graded as quiescent or healing. Both diffuse and local diseases structurally resemble chronic inflam-

matory and dystrophic conditions of bone and may accordingly be attributed to infective and endocrinological factors, acting separately or in combination.

Extensive biochemical examinations of blood and urine were carried out in 18 otosclerotics by Cassano.[5] There was a considerable excretion of 17-ketosteroids, a generally low dynamic action of proteins, a tendency to low blood sugar, intense sensitivity to insulin, and blood protein values and mineral metabolism within normal limits. There appears to be an isolated hyperproduction of adrenocortical androgens. The adrenal cortex plays a part in osteogenesis and bone nutrition (e. g., Cushing's disease), and the nitrogenous androgenic hormones stimulate enchondral osteogenesis particularly, which is important in that the periotic bone is of cartilaginous origin. The complex of metabolic disorders is related to predominant diencephalic changes. A diencephalohypophyseal pathogenesis has been suggested for Paget's disease, which shows some general histological analogies with otosclerosis.

The DL difference test, a measure of the degree of recruitment present in the operative ear, was administered to 55 preoperative fenestration cases. The prediction of the postoperative level (six months) based on the test was slightly superior to the prediction based on bone-conduction audiometry (Shambaugh method). Jerger[6] claims that use of the recruitment phenomenon is an additional predictive method independent of bone and speech audiometry.

The general inhalation technique of ether and oxygen with induction by nitrous oxide and oxygen administered by the nasal-tracheal route with tight intrapharyngeal packing and the discrete use of electrocoagulation, as employed in 1422 fenestration cases without serious complications, was reported by Stubensey.[7] The hazard of explosion or fire was never encountered. This method was the easiest to control and provided good operating conditions. The use of thiopental (Pentothal) sodium followed by ether and oxygen made it possible to intubate earlier but it did not otherwise improve the anesthesia. The addition of curare to the thiopental may have made the intubation somewhat easier but it made control of the patient's movements more difficult during the maintenance of the anesthesia. Of the local techniques, the intravenous use of morphine is too dangerous and too difficult to control, but the use of local and supplementary meperidine (Demerol) was found the most satisfactory. In the so-called uncooperative patient, complete general inhalation anesthesia is advised. The most recent anesthetic technique, and in properly selected cases, appearing to be the most promising method, is as follows: Local infiltration is supplemented with meperidine given intravenously throughout the operation. These patients were sedated with pentobarbital (Nembutal) 3 grains (0.2 gm.), three hours before operation and pentobarbital, 3 grains, one hour preoperatively. Then 100 mg. of meperidine was administered intravenously, the dose depending on the size or condition of the patient.

Lamp[8] reports the advantages of using an isothermal and isotonic solution for irrigation when making a fenestra in the labyrinth. He prepared a solution containing diffusible electrolytes of the same concentration as perilymph and nondiffusible particles with the same osmotic pressure as perilymph. One liter of solution contained 9.94 gm. of sodium chloride, and 6.093 gm. of sucrose when used at the ear at 37.0 C is isotonic with perilymph. The use of this solution reduced the amount of postoperative vertigo and nystagmus; there was noted an earlier rise in hearing; the loudness balance test showed no recruitment.

Bauer[9] reports a one-stage closed fenestration technique using a free conjunctival graft to cover the fenestra. The graft is applied, raw surface down, spread carefully to prevent rolling, and tucked gently into the fenestra to promote adherence of the graft to the membranous labyrinth. A wide (4 mm.) rubber drain is inserted into the

cavity without touching the graft. The postauricular wound is closed. Tube and stitches are removed together on the seventh day.

Hall [10] reports on 36 fenestration revisions with 76% showing considerable improvement in hearing after two years. Histological studies showed a fistula closed by connective tissue, in the deeper layers of which bone cells were found. It is contended that closure of fistulae takes place not by in-growth of the fistula edges, but by the formation of connective tissue in an accumulation of serum or blood beneath the flap. Bone is later formed in the connective tissue.

Farrior, Bagby, and Thomas [11] use a calibrated Weber test to measure the type, degree, and duration of serous labyrinthitis following fenestration surgery. Surgical trauma or operative hemorrhage in the inner ear produces a primary depression in bone conduction. Secondary depression in bone conduction seen on the seventh to ninth postoperative day arises from true serous labyrinthitis as a result of irrigation, endosteal manipulation, flap trauma, etc. With recovery there follows an improvement in bone conduction to preoperative level or better. In less favorable cases a tertiary cicatricial labyrinthitis may result in loss for high tones, unsatisfactory return of hearing, or even terminal degenerative nerve deafness. In the calibrated Weber test the oscillator is used over the midforehead. The bone threshold for each speech frequency is first determined, and then the lateralization at 10 db. steps to 60 db. maximum is charted. Tests are done at the end of the first, second, and third weeks. Results vary from complete ipsilateralization to complete contralateralization.

Angeluscheff [12] claims that the solution to the problem of otosclerosis is to be found in the study of the biological effects of sound waves on the ear. Because of the effect of ultrasonic waves on collagenous and bony tissue, due to reflection of the waves in passing from one medium to an adjacent different medium, a tremendous impact is made on the hearing organ. The specific location of this effect is at the borderline where ultrasonic sound waves strike the interfaces between two different media and are reflected (i. e., at the fenestra).

Ménière's Disease

The diagnostic significance and treatment of dizziness is presented by McNally.[13] If a patient complains of dizziness, one must first exclude the possibility that it is a warning sign of imminent labyrinthine invasion in the course of middle ear suppuration, quiescent or active. Next, serious organic disease (cardiovascular, central nervous system lesions) should be ruled out (seen in 35% of 200 consecutive cases of dizziness). Labyrinthine hydrops occurs in 30% of cases, diagnosis of which is aided by cochlear signs of recruitment, lowered threshold of discomfort, and reduced intelligibility at higher intensities. Dizziness without cochlear signs or symptoms occurs in the remaining 35% (pseudoMénière's syndrome). The origin of positional nystagmus or of directional preponderance is not yet well enough established to be of great diagnostic significance. The treatment is symptomatic. Scopolamine (Hyoscine) and its compounds and antihistaminics are effective labyrinthine sedatives. Nicotinic acid and histamine are useful for their vasodilator property. The operation of choice for Ménière's syndrome, when the disease is unilateral, is labyrinthine ablation. If the disease is bilateral, a differential section of the vestibular portions of both eighth nerves should be considered in the hope of preserving some of the residual hearing. A case of Ménière's disease should be given a thorough trial of medical treatment before surgery is contemplated.

In an etiological analysis of 100 cases of Ménière's symptom-complex Gundrum [14] found 48 associated with head injury, 34 with allergy, 10 with infections, and 13 of unknown etiology. Routine cytological examination of postnasal secretions was found of value in diagnosis and treatment. Those

with eosinophiles were placed in the allergy group, although in 12 there was no allergic history or symptoms. Most responded to treatment of the allergy.

Early recognition of a labyrinthine hydrops is most important, according to Langenbeck,[15] because during the early stages the process is reversible. In the first stage the low tones drop considerably; bone conduction is down to the air-conduction level, and there is no recruitment present. As a rule, dizziness and hearing loss are dramatically reversed with immediate treatment, in which dehydration plays the most important part. Elimination of focal infection, allergy therapy, and dietary measures will then prevent recurrences. Langenbeck regards this first stage of pressure labyrinth as a conductive disease of the inner ear. In a later stage the audiogram flattens out and recruitment is present. The lesion is no longer reversible, but treatment might still prevent recurrences. Therapy would have little or no effect in a still later stage, when degeneration has resulted in steep high-tone loss, with recruitment.

Droller and Pemberton[16] examined 476 elderly people and report on the incidence, causes, and effects of vertigo. Vertigo was found in 47.0% of men and in 61.2% of women. In the majority vertigo appeared to be closely associated with arteriosclerosis. In a small number a drop in blood pressure on changing from the supine to the upright position brought on an attack.

Wodak[17] classified the symptoms in 171 cases of aural vertigo. Rotatory vertigo (25%), unsteadiness in walking (18%), deafness (67.6%), tinnitus (47.2%), hyper-or-hypoexcitability of the vestibular apparatus (70%), triad of vertigo, tinnitus, and deafness (41%), and triad symptoms with objective changes of the vestibular apparatus (24%). Vertigo of the vestibular apparatus without objective changes is usually of less than six months' duration. Vertigo may be considered as a symptom of vestibular irritation which may lead to objective lesions of the vestibular apparatus if the basic damaging cause exerts its influence for more than six months.

Welt[18] applied Revici's hypothesis to the study of various conditions associated with vertigo. Revici has shown that many pathological states have a similar abnormal metabolism. These states have two broad patterns: one produces a local alkalinity and the other produces a local acidity in the intercellular fluid surrounding the lesion. Revici has shown that lipids are valuable in influencing the pathological state and in restoring metabolic balance. Welt presents 44 cases of vertigo treated with alkaline or acid lipids in which 75%-80% were freed of dizziness.

The role of electrolytes was studied in 15 patients with active Ménière's disease subjected to two extremes of sodium concentration by Perlman, Goldinger, and Cales.[19] One group remained on a low-sodium diet with ammonium chloride and mercaptomerin (Thiomerin) administration, which resulted in a low serum sodium level. The others were given extra dietary salt, retained by administration of desoxycorticosterone. The Kepler water test was used for evaluation. No consistent effects on cochlear or vestibular function were noted in patients with a measured low serum sodium content, as random fluctuations in hearing continued and some acute attacks of vertigo were observed. No acute attacks of vertigo and no deterioration of cochlear function was produced in patients whose serum sodium control had risen sharply. The negative results do not imply that the induced changes in extracellular fluids are necessarily reflected in the composition of the labyrinthine fluids. Nor does it refute the possibility that the disease is related to disturbances in the labyrinthine fluids, particularly the endolymph. It merely indicates that present therapy directed at changing the electrolytes is probably ineffective.

Selling and DeWeese[20] state that several factors contribute to a practical approach to the management of Ménière's syndrome. These are (1) an accurate diagnosis; (2)

knowledge of one or more ways to control the acute vertiginous attack; (3) awareness of the fact that more than one method is available for control of continuing symptoms and that if results have not been satisfactory with the first method used, another should be tried; (4) keeping in mind that the embarrassment and psychological shock produced by recurrent attacks must not be minimized; (5) continued contact with the patient; (6) willingness to recognize failure of medical management and to substitute surgical treatment when necessary.

For the treatment of Ménière's disease Ruedi [21] describes streptomycin therapy with controlled depression of the vestibular apparatus. The treatment is terminated before there is total loss of function. Hearing should not be depressed at all. Depending on the weight of the patient, 1.5-3.0 gm. streptomycin was given for five to seven days; the course was repeated when necessary after four to six months.

Kennon [22] evaluates the procedures suggested for the surgical treatment of Ménière's disease. Intracranial severance of the vestibular nerve, as recommended by Dandy, gives the following results: Vertigo is relieved in every case; hearing is preserved in one-third of all cases; tinnitus is also improved in one-third of cases. However, facial paralysis occurred in 1.7%. Neither Portmann's operation on the saccus endolymphaticus nor Rosen's procedure of cutting the chorda tympani are reliable. Cervical sympathectomy, advocated by Passe, did not give the same good results in other hands. According to the author, destruction of the labyrinth by the Cawthorne-Mollison-Wright-Lempert-Day method is most useful. He suggests applying this procedure in unilateral Ménière's disease, while for bilateral cases the Dandy method is advised.

Krech and Harris [23] discuss resistant cases of Ménière's disease. With medical treatment vertigo is definitely improved in 85% of cases; tinnitus in 50% of cases; and hearing in 20% of cases. Proper diet, dehydration, and potassium are valuable adjuncts of medical treatment. Surgical destruction of the vestibular endorgan is only rarely indicated; electrocoagulation is favored. Only such cases are selected for surgery where hearing is below the useful level. Prompt diagnosis is very important, because medical treatment has a good chance of success in the early stages, when the process is still reversible.

The use of intravenous procaine in the treatment of Ménière's disease is reported by Fowler.[24] He uses 250 cc. of 0.1% procaine in 5% glucose diluted with water and given in 8-10 minutes. Symptoms experienced during administration are listed. They were often annoying but not serious. The primary trigger factor in Ménière's disease is stress. Symptoms occur after long-continued aggravation in a susceptible individual. The use of procaine he considers a valuable tool to alleviate the symptoms of the moment and to obtain the confidence of the patient, so that proper psychiatric measures can be taken.

The direct application of ultrasonics to the bony labyrinth in the treatment of Ménière's disease is described by Arslan.[25] Results in seven cases are reported. These were examined for several months after the application. Vertiginous crises disappeared in all, and six showed distinct hearing improvement. Tinnitus disappeared or diminished considerably.

Atkinson [26] reports on a further group of 105 cases of Ménière's disease which he has treated along nutritional lines. Patients in the age group in which Ménière's syndrome commonly arises, 50 to 60, require a high-protein, high-vitamin, moderate-to-low-carbohydrate and low-fat diet. If patients with Ménière's syndrome are to be restored to full health and efficiency and not merely relieved of their attacks, insistence on such a diet is essential.

Surgical intervention in three severe Ménière's cases was performed by Menzio.[27] A small amount of fibrin sponge was pressed through an opening in the horizontal canal toward the ampulla without damaging the

membranous canal. Subsequent examinations showed more marked hypacusis, complete suppression of the function of the lateral semicircular canal, and a less intense postoperative vertigo as compared with other surgical methods.

Passe [28] reported the results of stellectomy and upper dorsal sympathectomy in a large number of cases of Ménière's disease. In 62% this surgery stopped the attacks of vertigo permanently, and in 35% of the cases gave relief. Part of this effect seems to be neurovascular, since those patients who also had migraine were relieved from any further headaches after sympathectomy. Preoperatively one must obtain improvement by injection of the stellate ganglion with procaine. The results of stellectomy were slightly more satisfactory than those of upper dorsal sympathectomy. The former operation involves section of the first thoracic white ramus communicans, causing Horner's syndrome, which is a disadvantage. Either operation resulted in substantially improved hearing in 31% and slight improvement in 32%. Tinnitus was relieved in 22% and reduced in 45%. Passe reported further that cases of nerve deafness which responded to procaine should have sympathectomy. Improvement of 10 db. and over was obtained in 24 of 32 such cases.

Complete psychiatric and psychological examinations were given to 40 patients with Ménière's disease by Zeckel.[29] Psychogenic factors and adrenogenic stimuli exert a great influence on the circulatory system and slow down labyrinthine circulation. Prior to Ménière's attack, blood sludge forms in the conjunctival vessels. Once lumping of red cells occurs in the smaller vessels, the predisposing condition for hydrops of the labyrinth is established. Stimuli resulting in impairment of circulation and ultimately in hydrops of the labyrinth can be reversed through psychotherapy in some suitable cases.

Stimulation of the cervical sympathetic trunk produces a reduction of the cochlear microphonic potentials in 20 out of a series of 23 animals (Seymour and Tappin [30]). The histological findings indicate that this phenomenon is due to a diminution in secretion of endolymph of altered quality rather than to the effect of a lowered oxygen concentration on the organ of Corti. Points of similarity to the early features of Ménière's disease are indicated and a possible mechanism for the later findings in this disease are discussed. The sympathetic nervous system is involved in maintaining the inner ear in a physiologically normal state. Autonomic imbalance produces definite histological changes, and, it is suggested, this imbalance is a definite entity in the production of some disorders of the inner ear.

According to Burian and Hausler,[31] vasomotor dysfunctions play an important part as underlying conditions of Ménière's disease. Blood pressure determination and pulse count are sufficient, and diagnostically the effect of loading the circulation with water is more significant. The patient is requested to drink 1 liter of tea; caloric examination is performed before and after loading; in most patients with Ménière's disease a strong vagus influence combined with hyperactive caloric response was noted following loading. Authors believe that the underlying vasomotor dysfunction, as well as Ménière's disease itself, can as a rule be successfully treated by shifting the vegetative functions to the sympathetic side, through administration of adrenocortical hormones.

In a series of 100 cases of Ménière's disease reported by Moritz [32] the great majority presented irritation of the cervical sympathetics. Osteochondrosis or traumatic changes of the intervertebral foramina exert pressure on the sympathetic nerve fibers which pass through these foramina to the vertebral artery. The pressure results in stimulation which is referred upward along the vertebral artery and down along the sympathetic chain. This is the principal underlying cause of Ménière's disease. Vegetative lability, endocrine dysfunction, emo-

tional factors, and the vagotonic make-up of the subject are also of importance. The inner-ear arteries are end arteries, and the lack of collateral circulation is the reason that pathologic changes become irreversible in a short time. Therapy must be started early. Traction of the cervical spine is the best therapy. Stellate ganglion block, which would be indicated in theory, is of little help in actual clinical practice. Surgical intervention to open the foramina and relieve compression of the sympathetic fibers is also advisable.

The hypothesis that neurovascular disturbances in the inner ear can be brought on by autonomic imbalance is being used today as a basis of treatment of Ménière's disease, tinnitus, sudden deafness, and nerve deafness. Rambo, Wolff, and Freeman [33] conducted two series of experiments on monkeys to test the validity of this theory. In one, sympathetic dominance was produced, and in the second, parasympathetic dominance. Complete surgical denervation of the parasympathetic supply to the inner ear was done in one series and of the sympathetic supply in the second. Complete denervation of the sympathetic nerve supply to the inner ear produced no immediate alteration of any significance in the caliber of the labyrinthine vessels. After complete denervation of either the sympathetic or parasympathetic supply to the inner ear observations carried out over 10 days to two months failed to reveal any signs of damage to the sensory structures of the cochlea or labyrinth. Therefore no evidence was found to support the hypothesis that neurovascular disturbances in the inner ear can be brought about by autonomic imbalance.

Healthy subjects and subjects who had previously undergone unilateral labyrinthectomy for Ménière's disease were exposed to centripetal force on a human centrifuge by Graybiel and Niven.[34] Subjective estimation of the change of position of objects in the visual field were determined. No significant difference was found for normal persons and patients with one labyrinth. This finding agrees with the fact that persons with only one or without labyrinths can very well sense their position in space relative to the direction of gravitational acceleration.

Beickert and Wust [35] demonstrated that cervical ganglionic block causes ipsilateral vasodilatation and contralateral vasoconstriction in the labyrinth. No dizziness occurred, and it is concluded that whenever dizziness is present it must have other reasons. It is assumed that sensitivity and irritability of the vestibular apparatus are affected. Cervical ganglionic block was performed and followed by caloric stimulation (5 cc. of water at 17 C): the reaction was studied with respect to duration of latency as well as duration and frequency of nystagmus. There was hyposensitivity on the side of the ganglionic block, and hypersensitivity on the other side.

Acoustic Trauma

Sataloff [36] investigated the effect upon hearing acuity of prolonged exposure to a specific steady noise ranging in intensity between 90 and 120 db. Comprehensive, rigid criteria were used in 154 subjects over a five-year period. The intensity and frequency characteristic of the noise and duration of exposure were correlated with significant hearing changes. Ambient noise levels were evaluated, as was the effect of auditory fatigue, in order to obtain stabilized hearing thresholds. There is indication that certain steady jet-engine noise with intensity fluctuation between 90 and 120 db. and with sound energy predominately in the frequencies below 600 cycles is not hazardous to the hearing of exposed personnel. Measurable degrees of auditory fatigue may develop daily for many years without producing any significant hearing loss. None of the subjects studied showed a hearing loss for the 1024 and 2048 cycles and none showed more than 30 db. loss at 4096 and 8192.

According to Larsen,[37] the degree of hearing loss which the individual worker incurs

when exposed to noisy operations depends on many factors, some of which are (1) the duration of exposure; (2) the interval between the cessation of work and the hearing test, the deafness being dependent on partly reversible processes (marked deafness even with older workers may improve considerably); (3) the quality of the noise (high-pitched noise seems to be more injurious than low-pitched noise; loud noise interrupted several times a minute has much more injurious effect than a continuous noise of the same intensity; resonance and reflection are of great importance); (4) the vibration; (5) the worker's condition (constitution, previous ear disease, age, the use of ear defenders). Subjective symptoms encountered are tinnitus (52%), vertigo (0.4%), insomnia (11%), headache (19%), and nervousness (33%). Regression is present in all cases of traumatic deafness, whereas it is absent in all cases of presbycusis. Prophylaxis is extremely important in occupational deafness. General measures of prophylaxis are (1) reduction at the source of noise; (2) prevention of noise spreading from its origin; (3) isolation of noise; (4) abbreviation of periods of very noisy occupation by change of workers to a less noisy place or frequent rest periods in quiet surroundings; (5) preemployment examination combined with periodic control examinations. It will also be of great value if, by means of a predictive test, it is possible to eliminate the noise-susceptible individuals before acceptance for employment. Most predictive tests are based on auditory fatigue. Auditory fatigue may be defined as a reduction of the sensitivity of the organ of hearing arising in connection with a preceding exposure to sound (stimulation deafness adaptation, acoustic trauma, adaptation remainder, residual masking). Larsen distinguishes two different phenomenon, fatigue in a narrower sense and adaptation. Adaptation means a decline in excitability caused by the stimulation, quite apart from the existence of activity. Fatigue means a decline in activity caused by the previous activity of the organ—that means a difference in physiological character. The adaptation effect is greatest at the stimulating frequency, whereas the fatigue effect is greatest half an octave above. With adaptation there is no frequency specificity, whereas with fatigue it is greatest at 4000 cps. Adaptation is never accompanied by buzzing in the ear, while fatigue often is. The adaptation effect is of shorter duration than the stimulus, while the fatigue effect is of longer duration. Larsen does not think it possible to get an exact picture of the auditory fatigue by measuring the effect of fatigue at only one frequency, because the individual differences of fatigue do not disperse at the different frequencies in a consistent manner. Larsen warns that an experimental fatigue test can produce a permanent hearing impairment and that the influence of a pure tone at great intensity is much stronger than that of noise at almost the same intensity. The relationship between short-time auditory fatigue and permanent hearing loss as a result of prolonged intense stimulation is not as yet clear, thus limiting the value of predictive tests. The use of ear defenders is effective for protection, but the majority of workers find ear plugs a greater burden than the loss of hearing without them. As to individual prophylaxis, a reliable predictive test is not yet found, and, even if such a one will be found, the number of susceptible individuals probably will be too large—and the last help, ear plugs, are not popular among workers. Therefore, the problem is very difficult, and the best method is probably preemployment examinations combined with control examinations of the workers.

Hearing loss in drop-forge workers was evaluated by Fox.[38] Factors which influence the degree of hearing loss from harmful industrial noise are (1) intensity and loudness of the noise; (2) period of exposure; (3) frequency spectrum of the noises; (4) individual differences in susceptibility; (5) age of subject; (6) distance from source; (7) character of surroundings in which the

noise is produced; (8) previous and coexisting ear disease, and (9) position of ears with respect to sound waves. A study was made of 64 drop-forge workers who had filed claims for alleged hearing loss due to industrial noise. Analysis showed that intensity of the noises was sufficient to cause hearing loss among the exposed workers. Hearing loss was proportional to the length of employment. Some of the early patients had only a slight or moderate dip at 4000 cps. Moderate and severe losses revealed involvement of the speech zone range of various degrees, with greater losses in higher frequencies. Usually both ears were equally affected. Total loss was not found. Tinnitus was present in 42.

According to Fox,[39] preemployment hearing tests are essential in noisy occupations. Where it is desired to employ a worker who has a marked loss for pure tones, the worker should have speech tests given him as well. Workers with nerve-type losses should be most carefully placed and most frequently checked. Workers with preexisting conductive losses are better risks in noisy environments than those with normal hearing.

Goldner [40] presented a critical evaluation of deafness in 600 shipyard workers. The hearing values in 403 typical cases were averaged. Length of employment, except for prolonged periods, did not result in a direct proportionate greater loss with greater length of employment. Older workers were more susceptible to acoustic trauma. Workers who were more intimately connected with the noisy process, who worked in confined spaces, showed greater hearing losses. Burners and welders are subject to burns of the external canal and tympanic membranes and may have perforations without their knowledge. Workers with otitis media or perforations of the drumhead are more susceptible to acoustic trauma. The problem of prevention includes the recognizing of the hazard, preemployment examination and audiograms, improved industrial design and soundproofing, medical control of exposed workers, the exclusion of workers more susceptible to hearing loss, and the use of efficient hearing plugs.

According to Neuberger,[41] no diagnostic method exists to differentiate between deafness due to electric and acoustic trauma. An attempt is made to work out an audiometric method. Electricity can cause functional organic damage. It is impossible to predict when damage to the labyrinth will occur because the current travels in the direction of least resistance. Damage is partly transient with maximal vasodilation, and partly progressive, sometimes over a number of years and assuming the form of slow degeneration. Very often the hearing organ is the only one affected, and then it is difficult to locate the lesion in the center or in the periphery. The sensory epithelium of the organ of Corti shows no damage, and such a finding can be the basis of differential diagnosis from acoustic trauma. There is nuclear damage in the medulla. In another group of cases bleeding of the tympanic membrane, middle ear, and/or labyrinth may be observed, while the organ of Corti itself remains unimpaired; there will be no recruitment in these cases. Acute acoustic trauma may cause damage to drum and middle ear, or leave them intact and only interfere with hearing. Trauma involves the organ of Corti. The resulting deafness is at least in the beginning of end-organ type and combined with recruitment. This finding is of great diagnostic value in cases which are examined soon after the damage has occurred.

Drooker [42] presents a statistical and critical analysis of auditory defects bases upon study of 490 soldiers who sustained head injuries and acoustic trauma. In many instances of penetrating ear injury there was associated atresia of the canal in varying degrees and depths, depending on the deflection of the missile causing the injury. Many soldiers survived extensive destruction of the base of the petrous pyramid. Of all fractures followed by ear complaints, 40% were penetrating temporal region fractures. The threshold of hearing on the injured side

was elevated to between 40 and 50 db. in the speech range. Head injury without fracture resulted in hearing as defective as that due to injury with fracture. Tinnitus persisted in 85% for 9 to 10 months after the injury; the statistics indicate that a blow on the head resulted in a greater hearing defect at the 4000 cps level when there was no clinical evidence of fracture than when fracture was present. Blast perforations of the drumhead do not protect the cochlear from detonation injury. The 4096 cps tonal dip was characteristic of blast acoustic trauma and more so of head injury. Forty per cent of the soldiers reporting ear symptoms were exposed to the periphery of an explosive blast wave.

Hilding[43] suggests that the hearing dip at 4000 to 6000 cps, following acoustic trauma, is due to the spiral curvature of the cochlea. The fluid thrust starting at the stapes will impinge on the outer wall of the cochlea at a point 6-8 mm. further on. This is the site for frequency localization of the tones in question. The actual lesion may be a laceration of the outer attachments of the tectorial membrane.

The tectorial membrane is normally attached to the outer hair cells and cells of Hensen. The attachments are delicate and easily ruptured by trauma. Hilding[44] suggests that the outermost attachment yields first, i. e., cells of Hensen, so the normal drag of the tectorial membrane would be applied to the next attachment in order, i. e., the hairs of the hair cells. This, it is suggested, would give rise to tinnitus.

Covell[45] exposed guinea pigs and dogs to frequencies ranging from 0.5 to 100 kcps, at intensities of 138-165 db., and studied the histological changes in the organ of Corti. The intensity for which the very severe injuries were found was usually about 150 db. or higher. Rupture of the organ of Corti and its release from the basilar membrane was a common finding. Moderate injury consisted in loss or degeneration of external hair cells and sometimes internal hair cells, collapse of the tunnel of Corti, and degeneration of phalangeal and other supporting cells. Some additional changes were degeneration in the connective tissue of the limbus, loss of mesothelial cells, and degeneration in spinal ganglion cells and peripheral nerve fibers.

The ears of anesthetized guinea pigs were exposed to intense tones of 185, 545, 2000 or 8000 cps at sound pressures (at the eardrum) from 138 to 148 db. by Davis et al.[46] The electrical output was recorded by differential intracochlear electrodes before, during, and after the exposures. The injury caused by 8000 cps centers in the basal turn, by 2000 cps in the second turn, by 500 and 185 cps in the third and fourth turns of the cochlea. No single tone, at the intensities and durations employed, injured all of the cochlea. The destructiveness of a tone does not depend on its frequency.

Deafness

The causes of hearing loss in 2628 children are analyzed by Kinney.[47] These cases were selected after screening tests at school, and 367 children with no usable hearing are not included. Conduction deafness occurred in 64.2%, perception deafness in 35.8%. Almost one-third of the latter group had normal hearing in one ear. Otitis media was the cause in 68.4% of the conduction-loss subjects. Middle ear effusion shows an actual and relative increase. No definite cause could be found in half the cases with perception loss. Of the others, meningitis and measles accounted for 23.9% and 23.2% respectively; the remainder followed mumps. The measles virus is of greater importance than previously realized. Meningitis as a cause is increasing. Mumps, birth injury, cerebral palsy, skull fracture, poliomyelitis, and pertussis are other causes mentioned. It was noted that a hearing aid worn by a child in a partially damaged ear would increase progressive hearing loss when compared with the opposite ear.

Hearing impairment in 572 preschool-age children was studied by Pauls and Hardy.[48] They were seen because of serious communi-

cative problems. All were tested by galvanic skin-resistance audiometry. Hearing was normal in 116 children. This group included neurological and psychiatric cases and cases with delayed speech. Of the 456 with impaired hearing 18 had conductive lesions of moderate degree; the others had perceptive or mixed deafness. No child had a total hearing loss. Relatively few children are totally deaf but rather are functionally deaf because they have not had the opportunity to use their residual hearing. Children with a hearing loss down to 75 db. below normal can make use of audition as the major pathway for language comprehension, with visual stimuli as adjuncts. With early use of hearing aids children with mild to moderate hearing loss can learn to interpret language through audition alone. Of the 456 children, 41% had up to 60 db. loss; 32% had between 60 and 75 db. loss. Children with more than 75 db. loss (27%) must learn to communicate principally by use of vision, with audition as a limited adjunct. Treatment and training should be started at 18 to 30 months of age. The child between 2 and 5 is at his peak as a language-learning individual.

The cochleopalpebral reflex is reported by Neidlinger[49] as the most valuable test in infants, and might be applied after the first week of life. Directional hearing begins after three months of life. Failure of this ability to develop suggests hearing impairment. Another sign is failure to develop some measure of articulate communication in the second year of life. For grading of hearing impairment the author suggests the following criteria: 1. The child hears in a small room, but not in a large room. 2. He cannot hear outdoors, but hears indoors. 3. He does not hear conversation, but hears shouting. 4. He does not hear shouting. 5. He cannot hear, but talks. 6. Deaf-mute, i. e., he has no pattern to imitate.

Hudgins[50] reports on an experimental program to study the effectiveness of auditory training in a residential school for the deaf. First objective: How much can be achieved by use of the best hearing aid in children with minimal residual hearing? Second objective: A standard group hearing aid is compared with a specially designed group hearing aid. Eight pupils were selected, average age 11 years 11 months. Six of them were congenitally deaf; two had had meningitis; of the group five were profoundly deaf; the remaining three had 65 db. in the better ear. Use of a powerful group hearing aid during the entire school day proved very beneficial; these pupils made better progress than a control group which used standard hearing aids. Speech intelligibility was less improved than speech perception. Even profoundly deaf children were helped. Lipreading still rates first place in the education of these handicapped children. Modern hearing equipment should be powerful, without producing distortion, and provided with a regulator to protect against acoustic trauma.

The aim of auditory training should be to make the aurally handicapped child fit for entering a regular school instead of a school for the deaf at 6 years of age, according to Bangs.[51] Auditory training should be started as early as possible. Hearing aids have been fitted as young as 15 months of age. Parents should give full support to a home-training program. Hard of hearing children can be taught to listen and discriminate only if they get hearing through amplification all day long as does a normally hearing child.

Visual perception in deaf children was studied by Myklebust and Brutten.[52] The deaf children showed a marked inferiority in their accuracy of marble pattern reproduction and other tests. Etiological subtypes within the deaf group did not differ significantly. Deafness implies an alteration in the basic response modes of the organism, and this alteration includes actual disruption of normal visual perceptual organization.

Inner ear pathology following maternal rubella (five cases) was reported by Lindsay, Caruthers, Hemenway, and Harrison.[53] One case was known to have severe bilateral

deafness, and the ears showed a bilateral sacculo-cochlear type of pathology. The abnormalities in the saccule consisted of thickening and distortion of the wall and adhesions to the macula with corresponding areas of macular degeneration. In the cochlear duct the malformations of the tectorial membrane and Corti's organ were considered to be the explanation for the loss of function. The reduction in the extent of the stria vascularis is interpreted as an indication of the direct effect of the rubella virus, which in turn permitted a greater concentration of the virus irritant in the endolymph, resulting in injury to the developing structures within the cochlear duct and saccule.

On the basis of a questionnaire on maternal rubella and congenital deafness Hay [54] found that there were 358 completely deaf children in New Zealand. The causes of deafness were given as follows: maternal rubella 133 (37.1%), meningitis 31 (8.7%), hereditary deafness 19 (5.3%), otitis media 19 (5.3%), maternal mumps 2 (0.6%), congenital syphilis 1 (0.3%), Rh incompatibility 1 (0.3%), and unknown 152 (42.5%).

In 1945 Cogan described a syndrome consisting of nonsyphilitic interstitial keratitis associated with deafness. About 15 cases of this entity are reported in the literature. It occurs mostly in young adults. The condition is a combination of eye symptoms and Ménière's syndrome. Deafness develops rapidly, with subsequent subsidence of the vestibular symptoms, leaving behind diminished or completely absent vestibular functions. Loss of function is never recovered, whereas the hearing improves in 25% of cases. In the acute phase antibiotics, cortisone, and intravenous histamine have been tried without success. In a case reported by Oliner et al.[55] periarteritis nodosa developed after the acute phase of Cogan's syndrome. In analyzing other cases from the literature the authors come to the conclusion that there might be some etiologic connection between the Cogan syndrome and periarteritis nodosa.

Lavalle [56] reported that all cases of retinitis pigmentosa examined audiometrically had changes of auditory acuity. In the majority of cases low tones showed a deficit of 30-40 db., then rose gradually to a near-threshold peak. Between 4096 and 8192 the curve regularly presented an indentation. Another fall for frequencies above 8192 was present as a rule.

Scleroderma is still an obscure disease; the only certain thing about it is that it is a disease of connective tissue; almost every organ of the body may be affected. Only very few cases with hearing problems have been reported, most of them by dermatologists. Brandstetter and Koehler [57] report the case of a 37-year-old woman, who developed scleroderma in 1944, and by 1948, at the age of 32, noted for the first time impaired hearing. The audiogram showed a curve indicative of combined conductive and perceptive deafness, with a considerable air-bone gap. The curves were bilaterally symmetrical. The vestibularis was normal. X-ray of the temporal bone showed no pathologic changes. The authors feel that scleroderma affected the elastic components of the middle and inner ear. The strictly symmetrical curves and the thin, transparent drums seem to disprove otosclerosis.

Deafness and syphilis are reported by Portmann and Grimaldi [58] as being on the decline in Western Europe. In 1500 deaf persons a 2.6% incidence of syphilitic deafness was found, including 0.2% congenital syphilis, 1.6% late hereditary syphilis, and 0.8% acquired syphilis. Deafness arising suddenly due to neurolabyrinthitis of secondary syphilis is rare. The most frequent form of deafness due to acquired syphilis is the bilateral progressive variety in a case of long standing treated insufficiently or not at all. Such cases also show involvement of other cranial nerves (polyneuritis or polioencephalitis). Congenital syphilis occurs as early cases (dystrophic or not) presenting the diagnostic problem of deaf mutism, and late cases. The latter form is seen fairly often and corresponds to the

classical clinical picture. The deafness is usually of mixed type and shows great variability of development, especially when speech audiograms are considered. Modern serological tests rarely give a negative result, and development to total hearing loss is unfortunately frequent. Penicillin treatment in cases of deafness due to acquired syphilis sometimes gives spectacular results, but in heredity syphilis the results are often disappointing. Combined treatment with cortisone and penicillin is of no avail unless it can be started very early.

Grant [59] made a study of six children who had been deaf from three to seven years after meningitis. A pneumoencephalogram prior to surgery is indicated to select cases in which hearing improvement can be expected. In three cases better hearing followed the pneumoencephalogram, but the improvement was insufficient. Five out of six children were operated on; in two of them hearing returned to a useful level; a third one showed less improvement. The operation consisted of (1) release of subdural fluid; (2) opening of cystic subarachnoid formations; (3) dissection of adhesions around the eighth nerve; (4) mobilization of the anterior inferior cerebellar artery, to release pressure on the eighth nerve.

In most cases of allergy the skin, the gastrointestinal tract, or the respiratory mucosa are the site of penetration of the allergen, and the same shock organs produce the specific antibodies. It is not very well known that in many instances deafness is caused by middle and inner ear allergy. The middle ear is lined with mucous membrane of the respiratory type; the endolymphatic labyrinth is a skin derivative; this explains the frequent allergic reactions of these organs according to Shambaugh.[60] In the great majority of cases the etiology of hydrops of the labyrinth is unknown. Food or inhalant allergy could be proved only in 10% of cases. In all cases of hydrops of the labyrinth allergy should be suspected, especially in the presence of other evidence of allergy. History as well as elimination and reexposure tests are most informative; skin testing is secondary and unreliable. The therapeutic doses of antallergens required are minimal, and no increase is necessary once a good response has been obtained. There are three forms of allergic middle ear deafness: tubal occlusion with secretory otitis media; tubal occlusion with recurrent acute otitis media, and tubal occlusion with chronic otitis media. Mucoid chronic otitis media should always arouse suspicion of allergy, and an eosinophil count of the smear often clinches the diagnosis.

Cochlear block or the abrupt occurrence of deafness without preceding affections of the ear is reported by Carco [61] in six cases. Cold appeared to be the etiological factor in five of the cases. In one case the facial nerve was affected as well as the eighth. It is contended that allergic and vasomotor components are often involved in the causation.

The pathological effects of vertebral diseases (arthritis, arthrosis, and lesions of intervertebral disk) are reported by Arslan [62] as due to compression of the vertebral arteries or to irritation of the vertebral nerve. The syndrome may be due to inability of one of the two vertebral arteries to dilate immediately owing to arteriosclerotic changes (especially at the level of the epistropheus and atlas, where the artery forms a double curve). There is only a vertebral nerve in the lower segment of the artery. In its upper segment, corresponding to the sinuous portion preceding its entrance into the cranium, the vertebral nerve is replaced by another (upper) plexus, composed of branches originating either from the first and second cervical nerves or from fibrils arising from the superior cervical ganglion. Therefore an action on the anterior cervical sympathetic trunk should be capable of having effects on the vasomotor activity of the labyrinth. On the other hand, labyrinthine vessels are regulated by the circulation of the cerebral vessels, and the latter is known to respond

to vasoconstrictor impulses originating from the cervical sympathetic trunk.

Rheumatic diseases of the cervical spine involving the ear are reviewed by Laskiewicz.[63] Acute and chronic rheumatic arthritis of the cervical spine may affect the ear through the vertebral artery, the sympathetic system, and the parasympathetic nerves. In seven cases of cervical arthritis the patients complained of hypacusis, noises, and vertigo on head movements. There was transient nystagmus and diminished response to caloric stimulation. The paravertebral injection of 1% procaine to block the sympathetic and the cranial parasympathetic resulted in disappearance of the vertigo and a lessening of the noises.

Inner deafness may be related to anomalies of the cranium and cervical spine according to Gerlach.[64] Some cases of inner ear deafness also show signs of disturbance of the liquor circulation. Earlier trauma, inflammation, or focal infection might result in adhesions from arachnitis. There may be anomalies of the skull, especially the base: the angle of the clivus is lower; the posterior scala is smaller, and the tip of the petrous pyramid is slightly elevated. Acrocephalic inner ear deafness has been well known since 1926 (Ruttin). Such deafness is of end-organ type. The vestibularis is unchanged in the majority of cases. A disturbance of liquor circulation causes a change in endolymph pressure. Ganglion block and massive dehydration are advised and have given good results.

An anatomical study by Thonner[65] shows a hitherto unreported vascular supply existing from the internal maxillary artery to the inner ear through the petrosquamous and petrotympanic fissures in the glenoid fossa. Embryological evidence is presented for such a vascular connection. The question arises as to whether inner ear disturbances such as tinnitus, impaired hearing, and dizziness could possibly be due to interruption in nutrition to the capsule of the labyrinth in certain temporomandibular joint conditions.

Ronkin[66] describes the development of the internal pterygoid, tensor palati, and tensor tympani muscles, which have the same embryological origin and nerve supply, but each of which has a different function. Alteration of the bite will affect the tone of the internal pterygoid muscle, which in turn causes alteration of tone in the tensor tympani muscle, causing deafness and tinnitus. Repositioning of the mandible relieves these symptoms in a certain proportion of cases.

Heller and Lindenberg[67] believe that deafness of nonorganic origin, whether monaural or binaural, is common and can be correctly diagnosed by study of the entire patient and adequate testing of auditory function. Patients with a serviceable hearing organ and no cerebral disease may complain of deafness because of distorted, perverted, or rejected psychological interpretation of acoustic stimuli. Behavior during the interview may be a clue to the nonorganic origin of the deafness. Speech of normal intensity, inflection, and quality tends to contradict alleged bilateral deafness. Among the clinical hearing tests used are pure-tone and continuous-frequency audiometry with observation of the patient through a one-way vision window during the tests, repetition of audiograms at intervals of several days, the speech-to-noise ratio test of Doerfler and Stewart, response to spondee word stimuli, the Lombard test, the Stenger test, and the psychogalvanometer test of hearing by pure-tone audiometry. Motivation for deafness of nonorganic etiology must be uncovered by psychiatric investigation.

The significance of aphasia in otology is discussed by Stamm.[68] Aphasia has many varieties, depending on topographic location, extension, etiology, and stationary or progressive character of the brain lesion. Motor aphasia is rare in otology. It may be associated with encephalitis, malacia, or abscess localized in Broca's area. The patient is incapable of spontaneous speech or has difficulty in repeating words, although he understands what is said to him. Dysarthria, the heavy thick speech often associated with hemiplegia, depression followed by euphoria, paraphasic naming, and telegraphic speech

are typical. Sensory aphasia is caused by localized lesion of the temporparietal area. It is characterized clinically by amnesia, agnosia, and dysphasia. That the patient is usually not conscious of his deficiencies distinguishes it from motor aphasia. Vascular lesions, particularly partial thrombosis of the sylvian artery, are a common cause. A brain abscess involving the upper level of the sylvian fissure may produce sensory aphasia. Global aphasia with complete loss or disorder of speech understanding and expression may be confused with deaf mutism or mental deficiency. Functional tests may show at least traces of auditory activity. Amnesic aphasia, the most frequently seen by otologists, is an important diagnostic sign of a left temporal lobe abscess. The lesion involves the caudal portion of the left third convolution of the temporal lobe opposite the petrosa.

Tinnitus was found by Venters [69] in 16.5% of patients with aural disease. Suppurative and catarrhal otitis media accounted for half the cases of tinnitus, while otosclerosis, degenerative and other forms of internal ear deafness caused the other half. The incidence of tinnitus increases with emotional upset and fatigue. Tinnitus without deafness occurred in 7.5%. There is no effective treatment. Most persons are not greatly upset and learn to tolerate the noises. Kennedy [69] described the psychiatric aspects of tinnitus. There may be a depressive preoccupation with tinnitus or the symptom may be used as a psychoneurotic escape. It may accompany anxiety states and senile deterioration or be a symptom of hypochondria. Elithorn [69] described a selected group of cases who had a modified prefrontal leukotomy with mainly helpful results. Cawthorne [69] pointed out that tinnitus did not seriously worry many patients. It sometimes indicated the offending ear in Ménière's disease. He warned against labyrinth and eighth nerve operation for tinnitus.

Heller and Bergman [70] report on tinnitus in normally hearing persons. Audible tinnitus was experienced by 94% of 80 apparently normally hearing adults when placed in a testing situation having an ambient noise level no greater than 18 db. Of 100 unselected patients with deafness, 73% described tinnitus as a symptom. It appears that tinnitus is present constantly but is masked by the ambient noise which floods our environment. This ambient noise level for ordinary quiet living conditions usually exceeds 35 db. and apparently is of sufficient intensity to mask physiological tinnitus which remains subaudible. Tinnitus, which is subaudible, may be a physiological phenomenon in an intact auditory apparatus. Tinnitus, then, will not be eliminated by any treatment but at best can only become subaudible.

When an ear is stimulated with a pure tone of a certain frequency and intensity, some persons may perceive an extra tone called an idiotone, in addition to the stimulus tone, according to Flottorp.[71] The perception may be in a form of beats, roughness, or distinct double tones (diplacusis monauralis dysharmonica), and it may be followed by a more or less rapidly decaying aftertone after the stimulus tone has been turned off (diplacusis monauralis echoica). The pitch of this transient tinnitus is nearly unaltered by the various frequencies that are used as eliciting stimuli. The frequency corresponding to the idiotone is characterized by an inability to elicit this idiotone for any intensity. This frequency is extremely stable. The influence of various aspects of the stimulus tone upon the evocation and the character of the idiotone are described. The idiophone effect cannot be used to support a theory of high-tuned circuits in the cochlea, but rather it points to some kind of time-consuming process, such as biochemical action in sensory elements and nerve cells.

Cawthorne and Harvey [72] compare hearing for pure tones and for speech. In pure conductive deafness there is such a close correlation between hearing for pure tones and for speech that it is possible to predict hearing for speech from the pure-tone audiogram. In conductive deafness with reduced

cochlear response (mixed deafness) there is a definite relationship between the hearing for pure tones and for speech, but it is not so close as is the case for conductive deafness. In perceptive deafness the ability to understand speech often bears little relationship to the hearing for pure tones. It is particularly unreliable in end-organ deafness (Ménière's disease), and it is in such cases that the loudness recruitment phenomenon is present. A similar pattern is noted in traumatic deafness, where again the lesion is in the end-organ. The disparity between the hearing loss for speech and for pure tones is not always so noticeable, though in the majority of cases the ability to understand speech levels above threshold does not run quite parallel with the normal, and in some cases the pattern is similar to that found in end-organ deafness. In cases of otosclerosis, the speech curve lies parallel to the normal. After the fenestration operation for the relief of deafness due to otosclerosis, the speech curve nearly always retains this relationship, and the shift of the curve nearer to the normal in favorable cases corresponds very closely to the improvement in hearing for pure tones. Monosyllabic word lists were superior to the spondee word list because the latter could be memorized with ease, thus making them unsuitable for repeated testing. It was sometimes found that with repeated tests the patient improved his articulation score even though different word lists were used to exclude memorizing. In such cases the best score represented the true auditory acuity of the patient.

Dix and Hood [73] review the loudness recruitment phenomenon. They particularly stress the difficulties which appear to have risen from the neglect of certain physiological principles concerned with the technique of this test. They point out that recruitment does sometimes occur in cases of eighth nerve neuroma (8%). This in no way invalidates the fact that in deafness due to tumors of the eighth nerve loudness recruitment is characteristically absent. The explanation for the exceptions is that the occurrence of recruitment is connected with hair-cell changes resulting from occlusion of the cochlear vascular supply. If the cochlear blood supply is restored, then loudness recruitment may disappear. The deafness in Ménière's disease is invariably attributed to hair-cell disease. With regard to the technique of the tests, the physiological principles of the loudness recruitment phenomenon make it essential to use a technique with short, intermittent stimuli. Other methods using longer stimuli may cause the recruitment phenomenon to be obscured by pathological adaptation. Loudness recruitment when it is present in end-organ disease can only be elicited by tones of short duration. Dix and Hood stress the use of short tones with a subject wearing a pair of telephone receivers, each supplied by a separate audiometer with independent adjustment of intensity. The frequency of the sound stimulus is the same in each receiver, and the tones are applied alternately from one ear to the other. The loudness recruitment phenomenon is manifested as a loss of sensitivity at low-intensity levels, which is fully restored at high-intensity levels. This nonlinear hearing loss, although the most commonly recognized, is only one aspect of a rather complex disorder, of which the other important characteristic is the "relapse" phenomenon. "Relapse" is in effect the reverse of loudness recruitment. It occurs in response to sustained auditory stimulation and manifests itself as abnormally great subjective loudness loss which can be very considerable and result in a matter of a few seconds in the complete abolition of sensation in the presence of what was initially a very audible stimulus. To summarize: First, in the testing of the loudness recruitment phenomenon, no matter what the techniques may be, the application of sustained tones should at all times be avoided. Secondly, serious consideration should be given to the matter of the total interruption in pure-tone audiometers. In this respect, it would appear that the build-

up time should be as short as possible, sufficient only to avoid the click which arises from a too rapid build-up of the tone.

Walsh [74] states that speech audiometry is within the means of the average practicing otologist. Speech audiometry affords the otologist an understanding of his patient's ability to hear speech not only at threshold but at the superthreshold intensities of everyday life. Speech audiometry is more satisfactory than the pure-tone audiogram as a means of predicting the possible results of the fenestration operation and in determining when a hearing aid will be of value. Speech audiometry affords an added test in making a differential diagnosis in cases of Ménière's disease, and in lesions of the eighth nerve proximal to the cochlea. On a speech audiogram the articulation function is a curve of the listener's responses to speech testing. This curve varies with the test material used. The curve for easier material is steeper. Threshold of intelligibility is that intensity at which the subject hears and repeats correctly 50% of the test material. It is important when recording the results of speech tests to indicate what test materials were used and conditions under which the test was performed. A discrimination score is the percentage of material correctly heard at an intensity above which no further improvement occurs. The average percentage of PB words heard at the levels of faint—55 db., normal—75 db., and loud—85 db. speech is the social adequacy index of hearing (SAI). The SAI is determined by averaging the discrimination scores at which the articulation function crosses the three vertical lines drawn at 55, 70, and 85 db.

King [75] calls attention to some of the imperfections of free field voice tests. Some of the weaknesses of that test are (1) the failure to standardize the technique of giving the test; (2) the inability to control absolutely the pitch and intensity of a whisper; (3) the lack of control of ambient noise; (4) the different acoustic properties of test rooms. He deplores the use of this method to assess the degree of hearing impairment. It is likely that at some time in the future, some form of speech audiometry will be used as the basis for hearing standards, and this will be the ideal.

Van Dishoeck [76] attempts to define a number of audiological observations which have been subject to discrepant views. Fatigue refers to the effect of stimulation with high intensities and of relatively long duration. It is characterized by a threshold rise mainly for higher frequencies than the frequency of the stimulating tone. The term fatigue should not be used for the after-effect in the first milliseconds of a normal excitation or for the loudness decrease during stimulation with tones of nontraumatizing intensities. Initial adaptation and prestimulatory adaption are different processes, and this fact should be expressed by distinguishing them as initial adjustment and loudness-adaptation. Initial adjustment in the first split second is the expression of the adjustment of the ear to a certain intensity according to the Weber-Fechner law, as masking is another expression of the same process. Thus what is determined as initial adaptation is the same as residual adjustment of masking. Prestimulatory adaptation can be compared with the adaptation of the eye. It is a loudness adaptation without any insensitivity for smaller intensities other than during the first split second necessary for readjustment of the sense organ. To avoid confusion with the clinical threshold-rising which means a partial deafness, this initial insensitivity for smaller intensities during adjustment should not be called a threshold-rising.

Hearing acuity for voice signals against a background of noise was assessed by Simonton and Hedgecock.[77] Both the type of hearing loss and the threshold level are necessary for an estimate of the ability to discriminate in noise. Middle ear deafness does not reduce discrimination of speech in environments of noise, whereas perception deafness results in moderate loss of discrimination in noise. In unilateral cochlear deafness, distortion due to recruitment in the

poorer ear did not affect discrimination scores for the better ear either in quiet or in noise. A favorable ratio of intensity between signal and noise is necessary for efficient hearing in noise.

Audiometric measurements of the Gellé and Runge tests were made by Kietz and Zangemeister.[78] In the Gellé test air pressure of 40 mm. Hg was exerted on the drum. In the Runge test the external meatus was filled with water. The Gellé test reduces the air conduction curve below 1000 cps; the Runge test decreases air conduction above 1000 cps., and increases bone conduction below 1000 cps. With perception deafness the results are exactly the same. In the presence of advanced stapes fixation, however, the two tests are negative (they do not produce any changes). The authors conclude that otosclerotic patients hear by skull conduction and do not show any real air conduction. In cases of advanced otosclerosis, it is impossible to mask one ear by air conduction without stimulating the other ear.

Van Egmond and Tolk [79] have constructed an instrument (otometer) which readily gives a clear and complete impression of your patient's hearing organ and determines the characteristics of the hearing aid he needs. The choice of a commercial hearing aid can be made at once, provided their properties are known. The whole procedure lasts no longer than five minutes. The instrument is a master hearing aid with large continuous ranges of independent variations which overlap the most important acoustic properties of the available commercial hearing aids. It has the following properties: (1) two adjusters of the frequency response, for high and low tones separately; (2) quantitatively adjustable output level; (3) the amplified signal is presented to both ears at the same time with an adjustable loudness ratio (balance); (4) the instrument contains a noise generator the acoustical output level of which can be regulated in relation to the speech output level; (5) an automatic volume control is built in, which has a continuously variable limit level.

Reconstructive Surgery for Middle Ear Deafness

The modified radical mastoidectomy, according to Juers,[80] is indicated in cases of cholesteatoma secondary to an attic-retraction perforation, particularly if the size of the marginal perforation is such as to constitute a "bottleneck" to the egress of exfoliated epithelium. However, if the disease process has done its own exteriorization of the attic-antral area, then conservation treatment may suffice. Good preoperative hearing can be preserved when diseased tissue is removed, the tympanic cavity left intact and in communication with the Eustachian tube, and when the stapes is not fixed. Interruption of the ossicular chain will still permit a serviceable level of hearing. The matrix in the attic may be preserved after careful inspection under magnification so that no secondary pockets or invaginations will be overlooked. If the preoperative hearing is below the serviceable level, the ossicles should be inspected, and, if normal mobility is absent, the incus and head of the malleus should be removed. Improvement in hearing can be anticipated only if the stapes is mobile and good cochlear function is present. If the condition is neglected until cholesteatoma invades the middle ear, an irreversible conduction loss results and a complete radical mastoidectomy becomes necessary.

Moore [81] reports his results with the endaural modified radical mastoidectomy. Suitable cases are those with cholesteatoma involving the mastoid antrum and attic. Frequently the middle ear is sealed off and is functionally normal. If the middle ear is sealed off or can be sealed off surgically, a dry ear is virtually assured since there is no possibility of continued drainage from secondarily infected mucous membrane near the promontory, hypotympanic cells, or Eustachian tube. Of 18 patients operated on, all had intermittent discharge for one or more years, and 17 showed evidence of cholesteatoma at the time of operation. After all pathological tissue is removed, the ossicles are inspected. If normal they are not dis-

turbed. If involved the incus and head of the malleus are removed. This avoids crusting, and the middle ear is more easily sealed off by a plastic tympanomeatal flap. If the middle ear is sealed off by a cholesteatoma matrix, it is not necessary to remove the covering matrix if the cavity containing the matrix is exteriorized and made accessible. The Heath type of operation was done on two ideal ears with excellent results. In the others the Bondy type was done. In the entire series dry ears were obtained in 15 cases; hearing was improved in 7; there was no change in hearing in 5, and further hearing loss occurred in 5. Average improvement in hearing was 21.9%.

Versteegh [82] urges that one is not justified in postponing conservative radical mastoidectomy because of good hearing. Postponement of the operation in these cases will lead to irreparable damages in the middle ear, to which the incus appears to be the most susceptible.

Tympanoplasty improves the sound conduction in the middle ear damaged by chronic middle ear inflammation, according to Wullstein.[83] Its object is to construct a closed tympanic cavity, ventilated through the Eustachian tube, and a sound-conducting system. The improvement in hearing is about the same as obtained by fenestration in cases of ankylosis of the stapes; but, as both labyrinthine windows are open, a new system of sound pressure transformation to the oval window must be built up by the operation. This is done by means of tympanic membrane substitute with free skin graft, by which the remnants of the ossicles, mostly with columella effect of the stapes, are used, while at the same time the round window niche is opened and a sound protection is constructed for it behind the newly rebuilt tympanic membrane. It is of course necessary that the inflammation of the middle ear cavity be completely healed and that any possible labyrinthine complication be excluded. In most cases the plastic operation can be performed at the same time as the radical mastoid operation.

Cases of chronic ear suppuration with a conduction deafness of 50%-80% should have a fenestration combined with a radical mastoidectomy according to Kulakj [84]. The cavity and fenestrum are covered with a graft taken from the posterior surface of the pinna. A considerable increase in hearing was obtained.

Derlacki [85] reports on the repair of central perforations of the tympanic membrane. The entire margin of the perforation is carefully touched with a tiny, tightly wound cotton-tipped applicator which has been moistened with a saturated solution of trichloracetic acid. The acid is also applied over the epithelial surface for a distance of 0.5 to 1 mm. This is followed by a light insufflation of trisulfapyrimidines (trisulfonamides) and Sulzberger powder (iodine and boric acid). The entire drum is then covered with a flat cotton patch which had been moistened with 3 or 4 drops of Euthymol (Parke-Davis & Company). The moistened cotton is then tamped snugly against the drum. Two drops of Euthymol are then instilled twice daily by the patient. A few developed suppurative otitis media which required temporary cessation of treatment. In 131 cases treatment was complete and 75.6% were successfully closed. In nine cases perforation recurred but eight healed again. Closure was obtained in an average of 15 treatments.

Repair of extensive eardrum lacerations which occurred during fenestration operations are described by House.[86] The promontory is curetted and a full-thickness skin graft is placed over this region in such a way as to cover the eardrum opening. The procedure may also prove of value in closing both central and marginal perforations in selected cases of inactive chronic otitis media.

Most perforations can be made to heal in 14 to 45 days by patching with a tissue paper disc, reports Benton.[87] This disc is held against the flat open end of a hollow metal rod and rubber tube by suction from the operator's mouth and is released in position by blowing gently.

Nielsen [88] used prostheses made of polyethylene in seven patients with perforations

in the drum with good results. The prosthesis has a small valve in the center of the concavity which facilitates its application and removal. It can remain in place several weeks without causing irritation.

Henner and Tamari [89] report five cases of "silent" unsuspected cholesteatoma. Repeated attic discharges may simulate an external otitis. A normal-appearing drumhead does not exclude the presence of a cholesteatoma. Attic perforation in acute middle ear infections is associated with cholesteatoma. Cholesteatoma in cases of intact-appearing drumheads or small perforations should always be treated surgically and the matrix removed completely.

Hughes and McKenzie [90] found cholesteatoma in 28 out of 41 cases of chronic otorrhea recommended for mastoidectomy. In none of these was a cholesteatoma diagnosed before operation, and exploration of the mastoid was advised simply because it was thought to be infected. The authors studied the hearing in 50 cases who had had a modified radical mastoidectomy, and found 26 had useful hearing and a loss of 35 db., or less, for the speech frequencies. None of the 50 cases had further loss of hearing as a result of the surgery. The otologist should operate more frequently when he suspects a cholesteatoma.

In the management of congenital ear canal atresia establishment of hearing should take precedence over plastic repair in all cases of bilateral congenital atresia according to House.[91] While awaiting surgery at age 3 to 4 every effort should be made to create awareness of sound in the child. At 10 to 15 months of age general amplification may be used for short periods daily. If the mastoid is well pneumatized on x-ray studies, a middle ear and ossicles probably exist. If sclerosis is revealed, it is less likely that a middle ear and ossicles exist. Determination of good cochlear nerve function is most important in selecting cases for surgery. Correction of atresia should always precede plastic reconstruction in order to restore hearing and speech as soon as possible, and also creation of a new canal through a plastic area might result in infection and loss of the newly created auricle. The original paper should be consulted for details of the surgery. In general, great care is taken to reach the antrum. In sclerotic cases the approach is superior to the mastoid region near the zygomatic root. The dural plate in the middle fossa is removed and used as a guide to reach the horizontal canal. The deformed ossicles are approached from behind through the epitympanum so as to avoid undue exposure of the middle ear region. The deformed ossicles are removed by rotating them anteriorly and superiorly. A thin full-thickness graft is placed over the smallest possible opening into the epitympanum to create a middle ear space and avoid scarring in the stapes region. Fenestration is immediately indicated if stapes fixation persists. It is indicated as a secondary procedure if the initial hearing improvement does not reach the practical level. Use of an acrylic mold seems imperative to prevent postoperative stenosis.

Concerning organization of exudate and pathogenesis of adhesive otitis media Ojala [92] concluded, from a histological study of 116 temporal bones (1) that inflammatory damage to tissues exceeds a certain minimum which is indicated by the presence of exudate containing fibrin; (2) that subsequent proliferative reaction of connective tissue is rapid and powerful, depending on resistance of the host and virulence of the infectious agent; (3) that drainage is impeded in clinical practice owing to the rupturing of the drum late or not at all. If the inflammation subsides rapidly and the conditions are favorable for repneumatization, the organized tissue usually atrophies and contracts without causing permanent hearing loss. If the inflammation is protracted or recurs frequently, the organized tissue in the middle ear cavities becomes denser and is transformed into permanent adhesions. The disease progresses if recurring inflammations cause recurring tissue proliferation and new adhesions.

The problem of the blue eardrum was reviewed by Johnston.[93] Suggested causes are (1) rupture of a hemorrhagic bleb in acute otitis media; (2) protrusion of the jugular bulb through a dehiscence in the hypotympanum; (3) chronic Eustachian tube obstruction, and (4) recurrent hemorrhages into the middle ear from an undetermined site in the mastoid, middle ear, or Eustachian tube. There is a persistent tendency of fluid to reaccumulate in the middle ear. If elimination of concurrent nose and throat disease and nasopharyngeal lymphoid tissue, repeated myringotomy, and inflation with thorough evacuation of the middle ear contents fail to alleviate the condition and mastoid x-rays show a pathological process, simple mastoidectomy is indicated. Johnston believes that somewhere in the middle ear cleft of these patients an area of varicosity or even hemangioma exists which repeatedly breaks down, permitting escape of fresh blood.

The arteries of the human middle ear were traced with a microscope through serial histologic sections by Nager and Nager.[94] For each of the arteries a detailed description is given of its course, anastomoses, and branches. A previously unrecognized branch of the anterior tympanic artery, for which the name of "ossicular artery" is proposed, was found to be the principal supply for the malleus and incus; the mucosal networks of these ossicles provide only a secondary supply. The stapes received blood from three arteries—the superficial petrosal, the superior and the inferior tympanic. None of the arteries in the stapedial region can be definitely identified with the stapedial artery of embryonic life. No vessels of any kind enter the thin parts of the stapes, the central region of the footplate and the shafts of the crura.

Cocaine injected into the tympanic cavity passes through the round window membrane into the labyrinth, causing a violent vertigo which may last for many hours. This phenomenon was used by Mayoux and Martin[95] to verify the permeability of the round window membrane. If the vertiginous reaction appears after 10-20 minutes, it is assumed that the membrane is normal. If the reaction is delayed or absent, it is highly probable that the membrane is thickened or that the niche of the round window is blocked. This method may be used in the study of conduction deafness caused by tubotympanic infections. Many cases of perception deafness are the result of an affection of the inner ear during attacks of a tubotympanic infection. A diminution of the permeability of the round window membrane may in these cases be the only evidence of a lesion in the tympanic cavity. The otosclerotic focus in the round window niche which may be responsible for a fenestration failure may easily be discovered by this method. It may also be of aid in the differential diagnosis of tinnitus. Disappearance of tinnitus immediately after injection may be a strong indication for tympanosympathectomy (peripheral origin).

In another paper Mayoux et al.[96] reported the effects of cocaine injections into the tympanum. In tubotympanic deafness the permeability of the round window had frequently diminished, but rarely disappeared altogether. In chronic ear suppurations the round window was impermeable. This explains the frequent involvement of the labyrinth in tubotympanic catarrh and its relative rarity in chronic otorrhea. If infiltration with cocaine gave relief in tinnitus, then section of the auditory nerve would effect a cure. If not, then a repercussive influence in the superior neurones has to be feared, which would make section of the nerve useless. Cocainization of the tympanum has made possible the experimental study of interdependence. By injection of cocaine, a fall in the auditory threshold was obtained, not only on the injected side, but also on the other side. It has sometimes been possible to break the interdependence, as the cocaine injection effected an improvement of the opposite ear by transiently interrupting the reflexes from the affected ear.

Harty [97] gives an account of the distribution of elastic tissue in the intratympanic articulations and in the associated muscles and tendons. The physiological function of this tissue in the ear is reviewed. Although senile deafness is predominantly of the perceptive variety, some conduction deafness is often found on closer examination. The pathology of the latter component may be explained by the changes in the elastic tissue of the joints and tendons.

Using fresh human temporal bones, Kobrak [98] made observations on the eardrum and ossicles during acoustic stimulation. The stapes vibrates with a slight phase difference from the eardrum around an axis vertical to the footplate. There is a gliding side motion in the incudostapedial articulation whenever the stapedius muscle contracts. In man a second type of stapes vibration around an axis parallel to the stapes footplate was observed. In rabbits the tensor tympani muscle contracts regularly in response to overthreshold stimuli. The muscle contraction is tetanic. Both muscles contract simultaneously. Acoustic vibrations of the muscle belly were seen during strong contractions. On observation of inner ear structures through a glass window it was seen that the fluid in the two scalae vibrate in opposite phase. Vibrations of the cochlear portion have a definite phase lag with the eardrum vibrations.

Toxic Labyrinthitis

The streptomycin group of drugs frequently damage the cochlear and vestibular mechanisms centrally, and possibly the peripheral end-organ also (Winston [99]). This damage is always bilateral. Streptomycin frequently damages the vestibular apparatus and rarely damages the cochlea, while dihydrostreptomycin frequently causes deafness as well as severe damage to the vestibular apparatus. The incidence of these complications is directly proportional to the size of the daily dose and to the number of days administered. With streptomycin deafness is usually preceded by tinnitus. It practically never occurs with daily doses of less than 3 gm. Deafness and vestibular damage are more abrupt with dihydrostreptomycin and may be delayed until after therapy has been discontinued. If therapy is discontinued when complications ensue, the hearing loss and vestibular dysfunction may improve. Audiometry and vestibular testing should be done before, during, and after treatment. Elevations in body temperature may give incongruous results in repeated caloric tests. While streptomycin appears to have a greater neurotoxic effect on immature rats than on adult rats and while it has been shown to cross the placenta, no neurotoxic effect upon the fetus has been reported. It would seem, therefore, that pregnancy does not contraindicate the use of streptomycin. In the treatment of Ménière's disease streptomycin renders paroxysms of vertigo less severe but does not eliminate them. Because it attacks the central vestibular mechanism and possibly other brain structures as well, it should not be used for this purpose.

It is difficult to decide on the basis of the series of cases presented by Anderson, Ohrt, and others [100] whether to recommend abandonment of dihydrostreptomycin for streptomycin. In some dihydrostreptomycin-treated series acoustic lesions have been demonstrated in from 5% up to as much as 80% in a single series (tuberculous meningitis and military tuberculosis) where half of the patients became totally deaf. In other series there has been found only moderate impairment of hearing at high frequencies, and in a single series no acoustic lesion whatever. The sizes of the single doses or the total dose give no exact clarification either, as severe loss of hearing to total deafness has been observed after a small dose (10 mg.), while in other instances several hundred grams have not influenced the hearing acuity. Similar variations have been shown with regard to the vestibular lesions.

Ormerod [101] reports on 93 patients, mostly children, who had recovered from tuberculous meningitis. Of these, 76 had strep-

tomycin and 17 dihydrostreptomycin. Of the total number, 56 had lost more than 70 db. of hearing, 12 had lost between 40 and 70 db. 4 had lost up to 40 db., and 21 had retained normal hearing. Those who had received dihydrostreptomycin had a higher degree of deafness. In almost every patient there was some diminution of the caloric response. The galvanic and Romberg tests were normal in all cases. The temporal bones of five cases dying of tuberculous meningitis showed marked tuberculous infiltration of the two divisions of the eighth nerve. Only two of these showed changes in the internal ear consisting of lymphoid cell infiltration.

Aschan [102] reported on 207 patients of all ages suffering from pulmonary tuberculosis who were treated with 0.5 gm. of dihydrostreptomycin daily, usually for 90 days. Audiograms were performed before, during, and after treatment, and no hearing loss due to dihydrostreptomycin was demonstrated in any patient. Vestibular symptoms did occur, but regressed after discontinuation of treatment. Adequate clinical response may be obtained with small doses, and these doses are safe from the point of view of hearing and vestibular function.

In six cases of pulmonary tuberculosis treated with dihydrostreptomycin or streptomycin the Békésy audiogram showed a hearing loss with complete recruitment (Liden [103]) supporting the view that damage due to streptomycin or dihydrostreptomycin primarily affects the end-organ. In 18 cases of tuberculous meningitis recruitment was absent in three patients with loss of hearing. These cases have been classed as retrocochlear lesions and have been interpreted as being due to meningitis as such. In the other cases of hearing loss with recruitment, streptomycin is considered to be the causative agent. Dihydrostreptomycin seems to have a greater toxic effect on the cochlear nerve than streptomycin. Of 10 cases treated with streptomycin, 4 developed hearing defects, and 7 damage of the vestibular organ. Corresponding figures for 10 cases treated with dihydrostreptomycin are 8 and 5.

The therapeutic and neurotoxic effects of streptomycin and dihydrostreptomycin in patients with tuberculosis were compared by Heck, Lynch, and Graves.[104] No distinct therapeutic advantage was found for either drug. The predilection for vestibular toxicity by streptomycin and auditory toxicity by dihydrostreptomycin was established. Such vestibular dysfunctions become manifest soon after the streptomycin is started, and are reversible, at least with a 120-day course of 1 gm. daily, on stopping the drug. The auditory dysfunctions become apparent only after three to four months of dihydrostreptomycin therapy, are progressive for several months, even though the drug is stopped, and are apparently irreversible. The factor of reversibility may hinge on the primitive, resistant nature of the vestibular apparatus as against the newer delicate auditory mechanism.

Patients with tuberculous meningitis reported by Greven [105] received varying amounts of dihydrostreptomycin by the intramuscular or intrathecal route. The total loss of vestibular function in children amounted to 67.6%, in adults to 61.2%. The total hearing loss was 15.3% and 6.1%, respectively. The toxic damage is closely related to the amount of dihydrostreptomycin used. There are individual degrees of response which are particularly dangerous because they cannot be foreseen; sometimes comparatively small amounts of the drug result in severe cochlear and vestibular dysfunction. Repeated audiograms are important to check on the toxic effect of the drug. As far as hearing is concerned, the toxic effect is not cumulative. With regard to vestibular function, action is cumulative; damage or even complete loss may occur early and suddenly, and is irreversible. Cochlear loss occurs first in the higher tone range and might then still be reversible. Once considerable loss has taken place in all frequencies, it is usually irreversible. Intrathecal application of dihydrostreptomy-

cin is more dangerous than intramuscular injection. Children below 5 years of age are more sensitive than other age groups. Animal experiments demonstrated that no central, nuclear vestibular damage had occurred and that the action of the drug is only peripheral. Autopsies in humans failed to produce histologic evidence of central lesion.

Toxic effects of dihydrostreptomycin upon the central vestibular mechanism of the cat were studied by Winston, Lewey, Parenteau, Spitz and Marden.[106] In vital staining experiments with trypan blue in 14 control cats there was no gross or microscopic evidence of staining of the central nervous system. Seven cats with ataxia but no nystagmus, after daily doses of 1 gm. dihydrostreptomycin for 14 days, were injected with trypan blue and killed. In only one of the brains was there evidence of staining, which was confined to the Purkinje cells of the cerebellar cortex and were most marked in the flocculus, dentate, and fastigial nuclei, and medial and lateral vestibular nuclei. A test group of six cats received 14 gm. of dihydrostreptomycin over 14 days (10-12 times the therapeutic dose for man). The brains showed pyknosis and shrinkage of nerve cells especially in and about the Purkinje cells of the flocculus and nodulus of the cerebellum, and also in the dentate and fastigial nuclei of the cerebellum. Slighter changes were noted in the lateral vestibular nucleus and ventral cochlear nuclei of the medulla. There were no pathological changes in the cerebral cortex of any of the cats.

Grizzle and Cutting[107] studied vestibular intoxication in cats, using streptomycin and dihydrostreptomycin in doses of 150 and 75 mg/kg., and in a mixture of 75 mg/kg. of each. The lower dose of streptomycin proportionately delays the onset of symptoms as compared to the larger dose, but that when toxic effects finally appeared, they were maximal. The smaller dose of dihydrostreptomycin produced no vestibular intoxication and the larger produced complete intoxication in only one cat and minimal symptoms in the three others. In the group receiving the mixture of both drugs, neurotoxicity occurred at about the same time as in the group on the lower dose of streptomycin, and it is therefore concluded that the vestibular neurotoxicity was due to the streptomycin component of the mixture.

The ototoxicity of dihydrostreptomycin and neomycin was studied in the cat by Hawkins and Lurie.[108] Intensive treatment with dihydrostreptomycin caused a disturbance of vestibular function associated with scattered degeneration of the hair cells of the ampullar cristae. The effect was later in onset and less severe than with streptomycin. The microphonic potentials of the cochlea were little affected, but the action potential component of the round window response was depressed. There was degeneration of the external hair cells, especially in the second and third turns; the internal hair cells were unchanged. The cochlea of the cat is less sensitive to dihydrostreptomycin, than to streptomycin, although in man the reverse is true. Neomycin in smaller doses had little or no effect on vestibular function, but it caused severe or complete degeneration of the hair cells of the organ of Corti, and corresponding loss of microphonic and nerve action potentials.

Escher and Rupp[109] investigated the metabolism of dihydrostreptomycin and of streptomycin (blood concentration, elimination, and relations to intoxication). The healthy rabbit eliminates dihydrostreptomycin very quickly. After damaging of the kidney with uranium nitrite in rabbits, an albuminuria but no elevation of nonprotein nitrogen occurred. Combined administration of dihydrostreptomycin and uranium produces an acute vestibular lesion and simultaneously a uremia. In these cases vitamin A prevents both the acute vestibular damage and the uremia. High doses of dihydrostreptomycin (400 mg/kg.) without uranium gave no toxic effects but high doses of streptomycin (400 mg/kg.) without uranium causes an acute vestibular intoxication and uremia. In rabbits given vitamin A neither

vestibular intoxication nor uremia could be observed.

Berger and Sachs [110] report a case of perceptive deafness following the use of tetanus antitoxin. Any patient with serum sickness showing a tendency to tinnitus, vertigo, or hearing impairment requires immediate therapy with epinephrine, antihistaminic, cortisone, intravenous histamine to counteract the impending nerve damage.

There exists an acute and a chronic type of carbon monoxide intoxication; Schullenberg [111] discusses the acute type. Perceptive deafness occurs and is usually bilateral, but the degree of hearing loss is symmetrical. Tinnitus is encountered only in the grave forms of carbon monoxide poisoning. There may be a certain degree of improvement, but then hearing is stabilized at a level below normal. Vestibular symptoms vary greatly; real vertigo is rare; true vestibular nystagmus is frequent. Discrepancies between caloric and rotatory nystagmus tests suggest central vestibular damage. The older the patient and the longer the exposure to gas, the worse will be the toxic damage. Histologically, degeneration of the central and peripheral pathways of the eighth nerve is observed, with other parts of the brain similarly affected.

Neomycin acts in vitro on many Gram-negative and Gram-positive bacteria, on Mycobacterium tuberculosis and on Proteus vulgaris. Clinically, neomycin has proved very effective against Pseudomonas aeruginosa, but was ineffective in tuberculosis. However, it is not only nephrotoxic, but also ototoxic. After a few days of application neomycin caused marked high-tone loss, the toxic effect becoming apparent about 10 days after discontinuation of medication. When neomycin was used over longer periods of time, total loss of hearing and complete loss of vestibular function occurred. In order to clarify the toxic action of neomycin, relatively small doses of the drug were administered to guinea pigs for one week, the total dose amounting to 4.8 gm. Ruedi and his associates [112] noted complete disappearance of the neuroepithelium of the organ of Corti; the vestibular portion showed no damage from such small doses.

Jatho [113] investigated the toxic effects of drugs on the functional performance of the vestibular system, particularly on nystagmus. The effect of alcohol and barbiturates produces a positional nystagmus, which occurs only in the erect position of body and head and serves as a useful sign of nystagmus preponderance. Scophedal (scopolamine plus opium) causes vertical nystagmus, which is independent of the position of body or head. Scophedal was given to children whose labyrinthine vestibular function had been damaged by streptomycin treatment. In these cases vertical nystagmus presents itself in five to nine minutes, and is not influenced by change of body position. The vertical nystagmus produced is exclusively downward. In the waking patient two forces counteract each other in the vertical plane; one force, the vestibular, pulls the bulbus upward; the other force, the cortical spontaneous, pulls the bulbus downward. In deep narcosis only the upward force will be effective if the vestibularis is intact. This gives a good indication of the level of scophedal anesthesia, because nystagmus develops as cortical depression sets in.

Vestibular Examinations

Nystagmus as a sign of central nervous system involvement is reviewed by Crosby.[114] Impulses originating in the peripheral vestibular apparatus pass over vestibular nerve fibers to the vestibular centers in the brain stem (lateral, medial, superior, and inferior vestibular nuclei) and directly to the cerebellum. Impulses from the medial and lateral vestibular nuclei cross the midline and pass up the medial longitudinal fasciculus to the abducens nucleus and then on up the medial longitudinal fasciculus to those portions of the oculomotor nucleus supplying the contralateral medial rectus muscle. Thus simultaneous contraction of the lateral rectus muscle of one eye and the medial rectus muscle of the other may be accom-

plished. Conjugate deviation of the eyes in the horizontal plane becomes possible. A smaller number of fibers from these vestibular nuclei ascend, without crossing, in the median longitudinal fasciculus to the ipsilateral abducens and parabducens nuclei, with the latter again relaying impulses forward to appropriate portions of the oculomotor complex. Such uncrossed fibers may be concerned with relaxation of the antagonistic muscles and this relaxation is provided for by reducing the flow of impulses over the related peripheral nerves. Irritative lesions in the lateral and medial vestibular nuclei will produce a nystagmus with its quick component toward the side of the lesion. Irritative lesions in the superior vestibular nucleus, and probably including also the upper part of the lateral vestibular nucleus, are indicated by vertical or oblique nystagmus. Rotary or irregular nystagmus suggests extensive involvement of the vestibular area of one side. The nystagmus in all these instances is due to an imbalance between the discharge of the vestibular centers on the two sides of the brain stem. Nystagmus may be due to other than vestibular stimuli. Watching telegraph poles while riding on a train produces an optokinetic nystagmus. Such responses depend on pathways through the occipital and preoccipital regions of the cortex. Frontal lobe lesions sometimes produce nystagmus. From the frontal cortex, the corticopontocerebellar system projects to the cerebellar hemispheres of both sides, although chiefly to the contralateral side, and to the cerebellar vermis, particularly to its contralateral portion. Among the regions in the vermis receiving such impulses are those overlying and connected with the nucleus fastigii. Irritative lesions in the cerebral cortex upset the functions of the nucleus fastigii in stabilizing cerebellar reflexes and produce a cerebellar type of nystagmus. Personality changes and other signs of frontal lobe involvement localize the lesion.

Anthony and Brown [115] review F. Kobrak's theory of nystagmus readiness or nystagmus preponderance, to which also the term nystagmophilia is applied. It is a centrally increased excitability of the vestibular pathways. Nystagmus readiness has been well known for a long time and it was demonstrated by Cawthorne, Fitzgerald, and Hallpike that temporal bone tumors influence and change the response pattern of induced nystagmus. Cold and hot (27 and 42 C) water irrigations, alternately in both ears, were used in order to find out whether there is a directional preponderance of induced nystagmus to the side of the lesion. A series of 20 patients was tested with temporal lobe diagnosis. Subsequently 14 underwent operation; in 10 cases temporal lobe tumors were found; in one case there was a large intracerebral hematoma; in the remaining 3 cases no space-occupying lesions were found. Directional preponderance may occur in the absence of cerebral tumors. The authors conclude that the preponderance test should always be combined with other tests. Visual field studies are more reliable than the preponderance test. The authors explored the diagnostic value of the recruitment test, the usefulness of which is by now commonly accepted.

Mygind [116] proposes a hypothesis which explains certain cases of vestibular phenomena in intracranial lesions as ordinary vestibular reflexes that have been liberated from their normal supranuclear, particularly cerebellar, inhibition. It is the same cerebellar mechanism which comes into play in vestibular compensation, Bechterew nystagmus (of which a vertical form is described), in nystagmus by labyrinthogenous cerebellar abscess, and many other phenomena.

Not only the vestibular but also the cochlear functions are subject to vegetative influences. Kobrak [117] suggests that the psychology of hearing, speech hearing, and musical hearing should receive greater attention in audiology. Monophone pure-tone audiometry by itself is insufficient. The complex influences of the vegetative nervous system on hearing are most clearly demon-

strated in damage due to trauma or shock. Present-day electrophysiologic experiments have not furnished information concerning tactile hearing below 18 frequencies, perception above 2000 frequencies, or the preacoustic phase just below the threshold. Pure-tone audiometry ascertains mainly acoustic responses; they are the basis of hearing efficiency which is more appropriately determined by speech-hearing or speech-understanding tests. Compensation from other sensory areas has been well known for a long time; adjustment of cochlear function is related to important higher centers.

For a long time it has been known that there exist interrelations and mutual influences between the vegetative (vagus) nervous system and the cochleovestibular system. Vegetative impulses also have a great influence on hearing. This may be due to a direct effect on the cochlear artery or on the secretion of lymph, with regard to its quantity as well as to its quality. Another factor are vegetative stimuli to the sacculus; according to Kobrak [118] the sacculus is somehow related to the hearing of complex, polyphonic tones. The third manner of vegetative action is through the middle ear muscles. All this leads to new problems in hearing and vestibular testing, because the results of tests will be greatly influenced by neuropsychic factors; in particular, noise audiometry comes much closer to hearing under everyday life conditions, than tests in a soundproof chamber. The same neurovegetative influences are present when loudness perception tests or air and bone-conduction tests at different (especially higher) intensities are performed. Only speech audiometry, particularly with noise interference, will give a true picture of hearing acuity.

The vestibular end-organ, states Mies,[119] is one of the terminal portions of the several known reflex arcs of the vegetative system. Vestibular stimuli spread widely, with distribution into nearly all of the reflex zones of the vegetative system. Very little attention has been paid to these reflexes. Observation of blood pressure, breathing, pulse rate, skin temperature, and responses to loading with fluid may give the examiner an insight into the vegetative make-up of the patient.

The vestibular reaction in many subjects shows a particular behavior: Deficient for normal values of stimulation, it recovered its strength with increasing the intensity of stimulation, until reaching the normal when the latter was further increased. According to Azzi, Giordano, and Spelta,[120] this response resembles auditory recruitment. Veit's method of caloric stimulation and increasing quantities of water was used. Most patients with this finding had suffered from head trauma with objective vestibular lesions, or had Ménière's disease. The phenomenon appears to be labyrinthine since it was not found in patients with a central or nerve lesion. In many cases an audiological recruitment was present. Some showed only the vestibular recruitment without any symptoms of cochlear damage. The authors express some theories to explain the phenomenon.

Cupulometry is a method of clinical examination consisting of the application of six impulses of 30, 60, and 15 degrees per second, respectively. Three rotations clockwise and three rotations anticlockwise are sufficient to determine the function of the semicircular canals. Van Egmond [121] considers cupulometry preferable to the classical method of Bárány because (1) the labyrinthine system is in a resting condition at the moment of the application of the impulse; (2) the stimulations in cupulometry are almost physiological (90 degrees per second maximally); (3) two different data (duration of nystagmus and duration of sensation) on the same system are measured; (4) several impulses of varying intensities are applied. The result obtained is in agreement with the predictions of the mechanical theory of the aperiodic pendulum, namely, that the duration of the reactions is proportional to the logarithm of the impulse.

Therefore it is an exponential function of time.

Hertz and Riskaer [122] describe a method for the electrical recording of nystagmus and voluntary eye movements and of the sensation of turning in well-defined accelerations. With the usual methods for the clinical examination of the vestibular system it is impossible to distinguish between lesions in various parts of the vestibular pathways: the peripheral vestibular organ, the central vestibular nuclei, the pathways between these and the ocular nuclei, the coordination system of ocular movements, and the sensation of turning. The authors aimed to investigate quantitatively the sensitivity of the ampullar cristae, the function of the vestibular nuclei, the coordination of eye movements, and the sensation and after-sensation of turning so that the disease in the four levels mentioned could be localized. They report some preliminary results which seemed to be of diagnostic value.

It is a well-known fact, reports Boenninghaus,[123] that after rotation the regular postrotatory nystagmus is followed by a second phase of nystagmus. The purpose of this study is to determine whether following calorization there also occurs a second phase of nystagmus, and whether it is comparable to the second phase of nystagmus which follows rotation. With the head in the least advantageous position, the ear canal was irrigated for 10 seconds, with 70 cc. of 17 C water, directed to the posterosuperior wall. After 30 seconds from beginning of irrigation, the head is brought into optimal position; now the canal is immediately irrigated with 100 cc. of 37 C water for 20 seconds. This is done to terminate quickly the cold-water reaction and thereby to study more easily the central reaction responsible for the second phase. This second phase is entirely a central reaction and should not be influenced at all by any change in the position of the head. The results of this test can be used only in conjunction with other otoneurologic findings.

One hundred normal subjects were examined by Petermann [124] for nystagmus preponderance; 20 cc. of water at 27 and 47 C was injected into the external auditory canal, in 20 seconds. Only one case showed nystagmus preponderance, which was traced to extraordinary vasolability. Even if no cause for nystagmus preponderance can be demonstrated, pathologic changes must be suspected.

In 83 patients with labyrinthitis Permin [125] compared caloric tests (using the Fitzgerald and Hallpike method but adding Frenzel's glasses), hearing tests, spontaneous nystagmus, and rotatory stimulation. The Fitzgerald-Hallpike caloric test did not appear to add any more information than did the determination of spontaneous nystagmus, positional nystagmus, and postrotatory nystagmus.

Labyrinth nystagmus evoked as a conditioned reflex was studied by Minkovskiy.[126] The sound of a Bárány noise box was used as a stimulating impulse in conjunction with rotation. The noise box was set in action 15 cm. from the ear while the subject was rotated for 10 seconds. The noise was stopped at the same moment as the rotation. In two sanguinic persons the nystagmus appeared at the mere sound of the noise box after 6-14 experiments. On interruption of these experiments the reflex disappeared, but it was easily restored by training. With a phlegmatic person there appeared only slow movements of the bulbi. The existence of a conditional labyrinth nystagmus proves that the latter is governed by the cerebral cortex, especially the fast component.

A new method for the clinical examination of nystagmus is described by Sullivan, Johnson, and Hodges.[127] They employ a small television camera together with a portable receiving unit. The simplicity of the method is stressed both for the examiner and for the patient; the former is freed from unstable devices, while the latter is unencumbered and at ease.

The behavior of endolabyrinthine pressure following intravenous administration of pilocarpine, atropine, epinephrine (Adrenalin), acetylcholine, neostigmine (Prostig-

mine) ergotamine, and histamine was studied by Filippi.[128] Pilocarpine has a hypertensive action due to a vascular mechanism and to a lesser extent to increase of spinal fluid pressure. Atropine is hypotensive probably owing to inhibition of secretion and filtration in the labyrinthine structures. Epinephrine always increases endolabyrinthine pressure due primarily to vasodilatation in the labyrinth and partially to spinal fluid pressure elevation. The action of epinephrine is reversed by ergotamine. Acetylcholine and ergotamine increase endolabyrinthine pressure due chiefly to increased spinal fluid pressure. Neostigmine occasionally produces the same effect, probably by vasodilatation. Histamine and anaphylactic shock are hypertensive, owing to increased spinal fluid pressure and possibly to altered vascular permeability in the labyrinth. Hypertensive drugs (epinephrine and acetylcholine) in high doses produce a marked hearing loss.

Anatomy and Physiology of the Ear

Rasmussen [129] reports the further course of the efferent cochlear fibers (originating from the superior olivary complex of the medulla) beyond the point he previously determined. This was determined in cats by means of the Marchi method and by silver impregnation of fragmented axons. The results clearly show that the efferent fibers (olivocochlear) traverse the vestibulocochlear anastomosis to join the cochlear nerve between the basal and the second turn of the cochlear nerve, enter into a spiral arrangement external to the spiral ganglion, and from here the fibers are distributed peripherally throughout all turns toward the organ of Corti. Owing perhaps to technical difficulties, degenerate fibers could not be traced into the organ itself, but instead could be followed only as far as a delicate spiral fasciculus situated near the margin of the osseous spiral lamina. Furthermore, it was found that not all spiral fibers are of olivocochlear origin, but that many are primary afferent fibers. A current conception of certain authors that these efferent fibers terminate along with cochlear afferent fibers on the internal hair cells is discussed in the light of personal observations.

Bocca [130] demonstrated the existence in the mammalian cochlea of two separate systems of nerve fibers in the lamina spiralis: (1) those originating in the spiral ganglion and distributed radially, and (2) a group independent of the ganglion and distributed spirally. In comparative anatomic studies of serial sections of rat and chicken embryos vestibular fibers innervating the cochlea were demonstrated. The fibers form part of the spiral system of fibers of the cochlea, which consist partially of fibers of vestibular origin and may contain a varying amount of autonomic fibers, probably of the postganglionic type, originating in scattered groups of autonomic cells or in isolated cells adjacent to the vestibular ganglion. Such fibers probably reach the cochlea with the vestibular fibers via the vestibulocochlear anastomosis. Vestibulocochlear fibers perceive the vibratory stimuli which may be regarded as a type of protopathic sensitivity as opposed to the epicritical sensitivity of the cochlear elements serving in tonal discriminations. It is necessary to distinguish between vestibular and cochlear hearing in the cochlea. The hypothesis of vestibular hearing seems to be in accord with many clinical as well as physical, physiologic, and phylogenetic considerations. Vestibular hearing in cases of total deafness can be considered the homologue of vibratory hearing in animals in which the epicritical sensory apparatus, cochlear nerve and its ganglion have not yet been developed. When the cochlear apparatus is sound, this vestibular hearing might well provide an auditory sensation with spatial qualities that possibly depends on the vibratory character of the low sounds.

The Portmanns,[131] using Weber's silver-staining method, were able to confirm Rasmussen's findings concerning the existence of efferent cochlear nerve fibers. These leave the vestibular nerve in which they descend from the contralateral oliva to the periphery

to enter the cochlea where they form Corti's anastomosis. The ultimate endings of these efferent fibers within the cochlea, showing the morphological characteristics of Weber's neurophysiological cycle, are considered to belong to the type of metaterminal apparatus. The efferent nature of these fibers is proved by topographical, morphological, and experimental facts.

Most surface epithelia have in their immediate neighborhood a rich capillary system for nutrition. Corti's organ has no such capillary system, and the only adjacent capillaries are found on the tympanic side of the basilar membrane below the tunnel of Corti, in the vas spirale. Engström and Wersall,[132] using electron microscopy, describe a rich system of minute filiform processes on the "outer" surface of the Hensen cells and the phalanx of the Deiter cells in the organ of Corti. Their possible functional importance for the nutrition of the hair cells is discussed.

Engström and Wersall,[133] using light microscopy and electron microscopy, describe the architecture of the supporting apparatus in the guinea pig cochlea. The structure of the supporting cells, their fibrillar structures and the relation between supporting cells and hair cells, and the relation of the nerve endings thereto are discussed. Even though the outer and inner hair cells present very marked structural differences, the nerve endings show remarkable similarities in the two cases. This study has an important bearing on understanding the neurophysiology of the hair cell.

Until recently very little attention has been paid to the tectorial membrane. Hilding [134] tried to determine the fibrillary structure and attachments of the tectorial membrane, using 130 fresh, nonfixed specimens. There exists an outer insertion of the tectorial membrane to Hensen's cells and another to the inner border cells. The tectorial membrane together with these two groups of cells forms a flexible arch with a limited range of movements. Underneath this flexible arch is another arch—the stiff arch of Held—containing the hair cells. The relation of these two arches to each other and the possible movements between them are of great significance. It had been assumed that with up-and-down movements of the basilar membrane the tectorial membrane and adjacent structures would move simultaneously, without any phase difference, but, according to Hilding, this assumption is no longer tenable. The movement of the tectorial membrane is transverse, sliding over the hair cells; the rate of waves and the pattern of their shapes proves to be the decisive factor. All the waves are characterized by sharp breaks; the one with its slope directed to the apex of the cochlea will determine the point of localization of a given tone.

Katsuki and Covell [135] describe the organ of Corti as studied under phase contrast microscopy. The hair cells are held in place by supporting cells and the reticular membrane. They are the most fragile components of the organ of Corti. They contain relatively clear ground substance of a more refractile type except for granules. The tectorial and basilar membranes are comprised of fibrils with intervening interfibrillar substance. The supporting function of the cells of Hensen is questioned. Peripheral nerve fibers and their endings are described for the organ of Corti. Two types of endings were observed for the inner hair cells, the significance of which is not known.

Pre- and postoperative hearing tests were done by Guild [136] on nearly 700 patients of W. E. Dandy operated on with eighth nerve section. In at least 75% the postoperative audiogram after partial sections of the nerve, either did not differ significantly from the preoperative test or showed only a slight recession of the upper limit of hearing (78 cases) by more than an octave from the preoperative level. Whenever there was a postoperative hearing loss, it was always for tones from the upper end of the audible range downwards; never was there a tonal island, or a raising of the lower tone limit.

Schuknecht and Wollner [137] performed partial section of the cochlear nerve in one ear of six cats. Auditory function was determined by the conditioned response method. After four to six weeks, the microphonic responses of the ears were recorded as well as a cortical test of auditory function. The ears were sectioned and graphically reconstructed. Ganglion cell counts were made along the spiral ganglion. The results were shown with the auditory function plotted on the anatomical frequency scale. Plotted beneath the audiogram was a chart showing ganglion cell density as a function of distance within the cochlea. Subtotal degeneration of the spiral ganglion occurred in four cats without corresponding change in the organ of Corti: the microphonic response to steady sinusoidal stimuli and clicks was normal. By selective section of cochlear nerve fibers restricted hearing losses were produced for both low and high tones. These findings support the concept that there is spatial representation in the spiral ganglion for low tones as well as for high tones. A large part of the spiral ganglion was sacrificed (60%-80%) in a particular region of the cochlea without creating a threshold elevation for frequencies having their fields of excitation in that region.

Hearing losses after experimental lesions in the basal coil of the cochlea were studied by Schuknecht and Sutton.[138] In each of six cats a lesion was made in the basal coil by directing a small needle through the round window membrane. The effect on auditory function was determined by the conditioned response method. The audiograms for all animals revealed abrupt hearing losses for the high frequencies. In five cats histological findings showed degenerative changes in the organ of Corti and spiral ganglion at the level of the basal turn. In one cat the spiral ganglion was involved, but the organ of Corti was normal. The results indicate that in the cat the points of maximum excitation for the frequencies 2000 to 14,000 cps are arranged in an orderly manner in the area 5 to 13.5 mm. from the basal end of the cochlea. It was also shown that with increasing intensity the spread of the excitatory process is less for high frequencies than for low frequencies.

Lindsay, Neff, Schuknecht, and Perlman [139] destroyed the saccus and endolymphatic duct in cats and demonstrated, histologically, three to nine months later that the organ of Corti and semicircular canals were completely intact. Functional testing by the conditioned reflex method also gave perfectly normal results. This shows that destruction of the endolymphatic duct has no effect on the structure and function of the labyrinth. Obliteration of the perilymphatic duct in another series of cats did not result in any histological or functional changes. In a series of guinea pigs the single efferent cochlear vein was interrupted. This did not cause impairment of function of the auditory and vestibular organ, suggesting that collateral efferent veins must exist.

Functional and histologic findings after obliteration of periotic duct and endolymphatic sac in sound-conditioned cats were examined by Schuknecht and Kimura.[140] Destruction of the endolymphatic sac failed to create cochlear dysfunction or histological changes in the membranous labyrinth. There is no definite proof that blocking the periotic duct causes dilatation of the endolymphatic system. In four cats the cochlear veins were interrupted and mechanically blocked with bone dust coincident to obliteration of the periotic ducts. Histological examination of these ears showed that drainage through the cochlear vein had been reestablished. Absence of acute vestibular and cochlear dysfunction following interruption of the cochlear vein must be interpreted to mean that venous drainage is adequately maintained through other vessels, possibly the communicating vein of the round window or veins accompanying the endolymphatic sac.

Wilkinson [141] reviews our knowledge of the nature of the hearing process and states that further knowledge will be derived from

deducting function from form and particularly by searching for evidence of design in nature. Important factors in conceiving of the cochlea as a resonator are the varying length of the basilar fibers, the varying distances at which they are placed from the windows, and the varying bulk of the spiral ligament which attaches them to the outer wall of the cochlea. With the use of a model of the cochlea, Wilkinson roughly illustrates Helmholtz's resonance theory and shows the possibilities of his "pitch-position" vibrations. The helicotrema plays an important role in providing a path of escape for such waves as are not completely absorbed by the basilar membrane and allows them to reach the round window; thus the basilar membrane does not absorb the whole energy contained in the sound waves transmitted to it. In a graphically recorded wave picture the differential and mimmation tones are represented by decided peaks rising above the level of the other vibrations. The summational tone, having a frequency equal to the sum of the constituent tones, is the highest in pitch, and so would be the first one to be partially absorbed by the membrane in the passage of the wave over its surface. The impulses giving rise to the beat tones are relatively large, that is, as big as the sum of the two wave impulses from which they are made up, because they have a frequency equal to the difference in frequencies of their constituent tones. They give periodic variations in intensity to the sound record, and so are capable of setting up localized responses on the basilar membrane which are perceived as tones of corresponding frequency when they become too rapid to be perceived as separate "beats." Any peaks in the sound record, if occurring at regular intervals, are capable of giving rise to a tone. Loudness depends on the number of nerve fibers stimulated and not on different intensities of stimulation of any particular fiber.

Some of the mechanical properties of the organ of Corti are discussed by Békésy.[142] The organ of Corti was observed in a lightly anesthetized guinea pig with a slit lamp. The relative stiffness of Reissner's membrane, tectorial membrane, reticular membrane, and Hensen cells was investigated with test needles. Stiffness of the reticular membrane and pillars of Corti was demonstrated. The stiff tissues of Corti's organ must be mechanically matched to vibrating energy of the perilymph. Corti's organ is well suited to act as a transformer for shearing forces. Observation of the membranes in Corti's organ showed only traveling waves during stimulation with a pure tone. For every given frequency there was one point along the basilar membrane where a transition in the vibration pattern occurred, and the more radial vibrations of Corti's organ near the stapes went over into longitudinal vibrations on the wave front facing the helicotrema. The place of transition moves with the frequency of the stimulus.

Zwislocki [143] states that the vibration pattern of the cochlear partition does not depend upon the path by which sound is transmitted to the cochlea (air or bone). By means of the hydrodynamic principle of continuity it has been shown that the vibration of the cochlear partition caused by a tone introduced into the cochlea either through the bony wall or through an artificial window can be cancelled by a tone transmitted through the oval window irrespective of the dynamic properties of the partition. Secondly, a general differential equation of hydrodynamic surface waves has been established, which shows that transverse waves are propagated along the cochlear partition every time there is a difference in pressure between the two sides. The wavelength is independent of the place at which the sound is introduced into the cochlea. Third, it has been deduced from Hamilton's principle of stationary energy that transverse waves in the cochlea must travel from the base toward the apex, irrespective of the location of the sound source.

Sinusoidal waves and nonsinusoidal periodic waves have a different loudness. According to Hennebert,[144] the threshold of

the latter waves is lower than that of sinusoidal waves in the range of low sound up to about 500 cps. The auditory area is formed of a series of elementary systems, superimposed and crenellated, and corresponding to a general biological property. The cochlear apparatus divides the frequency into unsharply delimited zones corresponding to cerebral localizations. In every zone, the precise discrimination of the frequencies depends on the frequency of the series of nerve impulses transmitted. These are distributed with an average frequency of 800 cps, reaching a maximum of 7000 cps at very strong intensities. In practice, however, the limit of 2000 is hardly ever surpassed.

Krejci and Bornschein [145] state that among the four bioelectric phenomena the research in cochlear microphonics is most promising with regard to the physiology of the cochlea as well as to future practical applications. The potentials are synchronous as to sound; they can, therefore, be recorded on tape, and the cochlea of different animals—like rats, rabbits, cats, guinea pigs—is used as a living microphone. Speech tests permitted establishment of the hearing frequency bands of different animals; tone localization was more closely pinpointed; ultrasonic radiation to the cochlear apex wipes out the hearing of low tones. Using pure-tone responses, cochleograms were taken. The electrical energy of the potentials is greater than the mechanical energy of the acoustic stimulus. The effect of anoxia, hypoxia, and of increased liquor pressure were investigated; they result in a decrease of the potentials, which usually proved reversible. The effect of acoustic trauma in guinea pigs was also investigated. Shortly after several gunshots had been fired, microphonic potentials were taken, and showed a loss of 10-30 db. around the 4000 frequency. The animals were killed at once, and degeneration of the outer hair cells of Deiters' cells was demonstrated. To produce chronic acoustic trauma, the animals were exposed for nine months to continuous heavy machine-shop noise. Young workers who just started to work in noise were also examined, and after nine months reexamined. In both humans and animals surprisingly similar impairment due to acoustic trauma was found. In man, the method of cochlear microphonics cannot as yet be used, because at this time it is not possible to produce a sufficient increase of the amplitude of potentials.

While cochlear microphonics were being recorded by means of an electrode placed in the round window of anesthetized cats breathing different mixtures of oxygen in nitrogen through a resuscitator, Wing et al.[146] collected samples of arterial blood anaerobically to evaluate the various degrees of hypoxia. Cochlear microphonics were reversibly reduced by (*a*) CO_2 within the limits used (5.2%-25% CO_2 in O_2); and (*b*) when arterial oxygen content was decreased from 14.1 vol. % to as low as 2.0 vol. % for as long as one-half hour. Complete recovery did not follow hypoxia below 2-3 vol. %. No more than eight to nine seconds was required for a single inhalation of room air to elevate microphonics already depressed by hypoxia. The generators of cochlear microphonics are thought to possess a very limited store of available energy for anaerobic function and are regarded as much more sensitive to hypoxia than has hitherto been believed.

The motions of the stapes were optically registered through the open vestibulum in 20 temporal bones by Wanderer.[147] The low tones of 10-50 cps were examined, the sound pressure varying from 300 to 90,000 dyn/cm^2. The stapes did not move at all at pressures below 300 cyn/cm^2. The footplate rotates around an axis which runs in a posterosuperior to anterioinferior direction. There are great deviations from the horizontal. The elasticity of the annular ligament has a marked influence on the amplitude of transmitted vibrations. Movement around the vertical axis occurs, but is of less importance.

According to Hilding,[148] up-and-down movements of the basilar membrane do not

cause stimulating movements between the tectorial membrane and the hair cells. This up-and-down movement is neutralized in the spanning zone of the tectorial membrane. Theoretically, when a bend occurs across the axis of the tectorial membrane and the organ of Corti, thus forming a slope directed toward the apex, the fibrils in the tectorial membrane are put on tension across this bend and stimulate the hair cells on the slope. As to frequency location, the basilar membrane is pictured as moving up and down as though hinged at the base for its full length from the base to the point of tone reception. Beyond this point it is held stationary by a double apical column of fluid which cannot vibrate at the given frequency.

In 10 B_1-avitaminotic rats, showing a marked decrease of pulse rate and of vestibular chronaxia, the cochlear potentials appeared to be normal (Schlander and Krejci [149]). Histological examination revealed no abnormalities in the cochleae. These findings support the view that disturbance of hearing in B_1-avitaminosis is due to a lesion of the eighth nerve and not of the cochlea.

In an important contribution by DeVries and Vrolijk [150] the phase relations between the microphonic crista effect of the three semicircular canals of the pigeon were found to reflect the second law of Ewald. Evidence was obtained that the crista effect has the same phase at both sides of the crista. The cochlea effect, recorded from the round window, has its maximum negative value at the moment the stapes has its maximum inward velocity for frequencies between 200 and 1000 per second. For lower frequencies the phase of the microphonic effect is ahead of the phase of the stapes.

In seven normal rabbits the vestibular nucleus of the medulla was stimulated by Kirstein.[151] After the experiments the animals were killed and the exact point at which the electrode had been located was ascertained in histologic slides. Homolateral jerking nystagmus was recorded upon application of alternating current—the eye moving in the direction of stimulation. If direct current is applied to the proximal end of the vestibular nerve, it will result in galvanic nystagmus of both eyes; a switch of the direction of current will change the direction of the nystagmus to the opposite side. Direct current stimulation of Deiters' nucleus will again cause bilateral nystagmus of the galvanic type, but no change of direction is observed after switching the current. The change of direction of the nystagmus upon switching the direction the current is linked to the peripheral portion of the vestibular nerve.

Kirstein [152] found that during caloric stimulation a rhythmic action current is discharged from the entire reticular substance. The direction of nystagmus changes the polarity of the discharge. The latent period before any discharge occurs varies in different animals. No action potentials were discharged from the triangular nucleus.

The influence of various unilateral cortical lesions upon the ocular reactions to 10 alternate clockwise and counterclockwise rotations was studied in dogs and cats by Wycis and Spiegel.[153] After occipital or frontal lobectomy the postrotatory nystagmus to the side operated on distinctly predominated over that to the normal side (directional preponderance). The change lasted from a few days to two weeks after complete elimination of the area striata, 5 to 10 days after frontal lobectomy. Superficial lesions of the parietal or temporal lobe had only a slight, if any, effect upon the postrotatory nystagmus, while deep-reaching lesions of the temporal lobe produced a definite directional preponderance, apparently due to injury of pathways connecting the area striata with the brain stem. After bilateral optic nerve section the duration of the postrotatory nystagmus was increased. After elimination of afferent impulses from the retinas unilateral occipital lobectomy failed to produce a directional preponderance, while unilateral frontal lobectomy was still able to induce a prevalence of the postrotational nystagmus to the side of the cere-

bral operation. Unilateral hemidecortication produced a much more prolonged directional preponderance than circumscribed lobectomies. An analysis of this difference suggests the participation of subcortical factors, besides the summated effect of the release of the vestibulo-ocular reflex arc from the influence of frontal and occipital cortical centers.

It has been found that the visual axes deviate in some individuals following vestibular stimulation by means of the Bárány turning test. The question arises whether this deviation results from the stimulation of the vestibular organs or whether it is due to something else. Hilding [154] found that the deviation of visual axes following the turning test as evidenced by the Maddox rod test does not appear to be a result of labyrinthine stimulation but rather it seems to be a manifestation of the accommodation-convergence or fusion reflexes, or both of them. It occurred in only about half the subjects. If this phenomenon were a result of labyrinthine stimulation, one would expect it to occur in normals each time the labyrinth is adequately stimulated.

Hilding [155] studied the minimal angular acceleration which is conspicuously perceptible, presumably due to deviations of the cupula. This time is about 0.5 degrees per second. The test was done by starting with one turn per minute and increasing the acceleration (or deceleration from a period of acceleration) until the subject correctly reported a turning sensation. However, when a subject is turning slowly without acceleration or deceleration, after having been subject to low rates of both, he seems to be unable to determine when the acceleration or deceleration has ceased. Near threshold a "gyratory illusion" similar to the optical illusion from ambiguous or unusual retinal signals seems to occur. Signals from the labyrinth similar in their ambiguousness seem to develop in the consciousness a definite sense of turning in one direction or another even when there is no change in acceleration.

In experiments with rabbits action potentials of the vestibular nucleus of the medulla were taken by Eckel [156] in the following manner: The cerebellum was aspirated, and noninsulated points of fine needle electrodes were placed into the triangular (cuneate) nucleus and Deiters' nucleus. The vibrations of the oscillator were recorded by means of a motion picture. The action potentials change with the deviation of the cupula. A utriculopetal deflection increases, while a utriculofugal deflection decreases the output of action potentials. This is the case during rotatory stimulation of short duration, 180 degrees in 2 seconds. In rotatory stimulation of a longer duration (90 degrees in 1 second) there will be no prerotatory stimulus; stopping of rotation is in itself the only stimulus. After rotation to the right the activity of the left side increases first; in the next 30 seconds the stimulus is equalized, and later on the right side takes over. The postrotatory nystagmus is directed to the side of increased activity. Deflection of the cupula plays a part in the direction of postrotatory nystagmus, which formerly was assumed to be central.

Head nystagmus in pigeons was registered by Van Eyck [157] by measuring the electrical effect of the neck muscles. Stimulation of one horizontal canal gives contractions and relaxations of only the contralateral muscles of the neck without reciprocal innervation of the muscles of the homolateral side (Sherrington). Extirpation shows that only ampullopetal movement of the endolymph in the horizontal canal can provoke a muscle reaction. The ampullofugal endolymph stream has no effect at all.

Capillaries of vestibular membranous labyrinth in the guinea pig were studied by Smith.[158] The macula acoustica of the utricle is supplied entirely by the anterior vestibular artery. The macula acoustica of the saccule receives its arterial supply from both anterior and posterior vestibular arteries. Arterioles to the saccule coil in the bone along the inferior wall of the vestibule, lateral to the macula, turn toward the macula

acoustica, pass over the cribriform opening for the saccular nerve, and enter the connective tissue with the myelinated fibers. The densest capillary networks are the subepithelial plexuses below the hair-cell areas. The capillary bed has the appearance of a flat, curved plate of radially directed anastomosing vessels. Its form and shape are adapted to the contour and size of the neuroepithelium. The arterioles enter near the center and the capillaries are brought together into venules at the periphery. The rest of the membranous labyrinth is surrounded by a loose capillary network except along the convex surface of the semicircular canals and the free membranous covering of the saccule, which is generally avascular.

The presence of very fine nerve fibers ending in the wall of labyrinthine vessels was demonstrated by Berendes and Schallock,[159] using a modified Bielschowsky silver-impregnation method.

The results of studying statoconia in man, rabbit and guinea pig by means of electron microscopy and x-ray crystallography are reported by Carlström, Engström, and Hjorth.[160] They are composed mainly of calcium carbonate in the form of calcite. Fish statoliths, however, were composed of aragonite, another crystalline form of calcium carbonate.

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Abstracts from Current Literature

Ear

Modern Management of Otitis Externa. J. W. McLaurin and T. P. Raggio, Eye, Ear, Nose & Throat Month. 35:641 (Oct.) 1956.

The authors outline the basic principles for treatment of otitis externa. Since there is no specific remedy, individualization in therapy is essential. Adequate time must be allotted each patient for adequate study. In the case of an acute exacerbation, relief of pain is essential, and analgesics should be used without hesitation. To control severe pain, x-ray therapy is recommended, giving one treatment only of 100 r.

The ear canal must be cleaned carefully, perferably manually, using tip suction. The canal is then wiped carefully with applicators moistened with 50% Cresatin or with grain alcohol. If the canal is too swollen or tender for suction or manual manipulation, it is irrigated with tap water. If the canal is not dry, it is then irrigated with grain alcohol and dried with compressed air. Compresses of Burow's solution, either full or half strength, are used for 24 hours when the canal is swollen, tender, and weeping.

Topical preparations are selected on the basis of cultures and sensitivity tests. These should be aqueous solutions with only one active agent, and with a pH that is neutral or slightly acid. The ear canal is filled with the solution several times daily for five minutes by the clock, and is then dried with cotton. The infection is usually under control after four days of topical application. Normal physiology of the ear canal is restored by the use of a hydrocortisone ointment or by dusting medication in a boric acid powder base.

By following these basic principles, usually 75% of the infections of the ear canal can be cleared satisfactorily. In the remainder of the cases, the predisposing cause involving either local or systemic processes must be discovered. Trauma from scratching is an outstanding cause, but now, with the help of hydrocortisone preparations, the problem has been simplified. Attention must be given to allergic, endocrine, and general dermatologic factors. Frequently there is a psychogenic factor. This may require psychiatric referral.

It may be possible to offer seasonal prophylaxis combating the loss of the normal protective oil of the ear canal in periods of persistent high temperature and humidity or from repeated wetting of the ear canal by frequent swimming or bathing. The authors use a silicone preparation obtained from the Dow Corning Corporation called Dow Corning 200 Fluid (viscosity 500). The pH is neutral or slightly acid, and in 165 patients no one proved sensitive to it. The solution is instilled in the ear canals about every third day during the time the ear is exposed to excessive moisture.

Jennes, Waterbury, Conn.

Heparin in the Management of Presbycusis. Emery I. Pick, Eye, Ear, Nose & Throat Month. 35:645 (Oct.) 1956.

Intravenous heparin is used for the treatment of presbycusis, both for prevention of further progress of the hearing loss and for relief of tinnitus. Forty-seven patients were given a weekly dose amounting to 300-400 mg. of heparin for a period of from three to five months. Heparin is an important factor in the metabolism of serum lipoproteins. Along with advancing age, there occurs an abnormal lipoprotein pattern, and heparin, which produces a reversal of this abnormal pattern, is present in decreasing amounts with advancing age. The author associates presbycusis with coronary, cerebral, and peripheral atherosclerosis which are also associated with the aging process and in the treatment of which heparin has been encouraging.

A series of 47 patients were treated with the average age in the mid 50's. Audiograms were taken at regular intervals, and, whereas no appreciable hearing improvement was noted, there was no further deterioration, either by bone or by air conduction. Follow-up audiograms three months and six months after termination of heparin therapy showed no further loss in any range. In the majority of patients, tinnitus was greatly diminished, and in some it disappeared completely. In several patients, tinnitus returned after termination of treatment.

ABSTRACTER'S COMMENT: This report is interesting, and such clinical investigation should be encouraged. Investigation of new modes of therapy must, however, be based upon the effective use of controls. The author's conclusions would be aided by the use of an additional group of patients selected in a similar fashion and given similar treatment with a placebo. This must be done with utmost discipline to prevent the patients from ascertaining whether they were in the treatment or in the control group.

JENNES, Waterbury, Conn.

RECENT ADVANCES IN THE MANAGEMENT OF DEAFNESS. HOUSTON L. BELL, Eye, Ear, Nose & Throat Month. 36:35 (Jan.) 1957.

In a relatively short article and in a very concise manner, the author presents the historical background relating to recent advances in the management of deafness. The modern otologist must have a broad knowledge of medical otolaryngology as well as the expected knowledge of modern surgical treatment. The treatment of secretory otitis media depends upon successful diagnosis and therapy of the underlying cause along with adequate treatment of the middle ear. The latter includes inflation, myringotomy, and occasionally the use of vinyl tubing through the myringotomy opening. Chronic suppurative otitis media includes perforations of the pars flaccida which are managed with the classical tympanoatticotomy without altering the tympanic cavity, and large marginal perforations of the pars tensa, which are treated by tympanoplastic procedures.

The relative advantages of both fenestration and stapes mobilization of hearing (defined as a 30 db. level or above) was achieved in 75% of the patients treated by fenestration and in 44% of the patients treated by stapes mobilization. Many patients also received considerable hearing gain from the stapes procedure, although they could not be included in the number of successful cases because the level of hearing did not reach 30 db. The author would not have subjected these patients to the fenestration operation.

Perforations of the tympanic membranes are treated by closure of the perforation by the Linn technique, or by the use of a split-thickness skin graft by the transmeatal route. In the former treatment the edges of the perforation are escharred with trichloracetic acid, after which a piece of cigarette paper moistened with glycerine is applied to the drum.

The ever-increasing attention to the causes, diagnosis, and prevention of deafness, and to the reparative surgical procedures for improvement of hearing are evidences of the concern of the otolaryngologist for the deafened. The metamorphosis of otolaryngology followed the introduction of chemotherapeutic agents and antibiotics, with the reduction in the incidence of mastoiditis and complications following ear infections. Over a period of years the field of otology has advanced to a position of prominence owing to its tremendous contributions to reparative and rehabilitative surgery.

JENNES, Waterbury, Conn.

SECRETORY OTITIS MEDIA. G. A. BATEMAN, J. Laryng. & Otol. 71:261-270 (April) 1957.

In this interesting article on secretory otitis Dr. Gordon Hoople is given due credit for focusing the attention of the otologist on a disorder of the ears described as far back as 1874, yet unrecognized at this time. Sixty-seven cases divided into three groups are reported. The first group comprises children with stuffy noses due to adenoids. Group 2 consists of subjects with deafness following otitic barotrauma. Group 3 consists of patients who suddenly become deaf in one or both ears after stuffiness of the nose. The symptoms and clinical diagnosis are well discussed, and emphasis is placed upon the fact that when first inspected the tympanic membrane appears slightly dark and indrawn. Occasionally a fluid level is seen. Treatment consists in complete evacuation of the fluid from the middle ear, either by needle aspiration or tympanotomy. These procedures are well described.

LEJEUNE and KEARBY, New Orleans.

ABNORMAL PATENCY OF THE EUSTACHIAN TUBE: SURGICAL TREATMENT. KINSEY M. SIMONTON. Laryngoscope 67:342 (April) 1957.

Abnormal patency of the Eustachian tube is an unusual but not rare condition. It can be recognized by marked autophony and loud swishing sounds when breathing through the nose.

Hearing is not usually affected, and there is no real tinnitus. The drum can often be seen to move with respiration.

The anatomy and physiology of the Eustachian tube are discussed particularly in relation to its opening and closing. Muscles and elastic fibers both play a part.

Causes of Eustachian tubal patency include adhesions about the pharyngeal orifice, scarring and atrophy of the tube due to irradiation, destructive lesions, and emaciation.

Nonsurgical treatment consists of applying irritants to the mouth of the tube. Surgical treatment, such as reported here, has not appeared in the literature. The technique and results are discussed in detail. Briefly, no ideal results (i. e., partial closure of the tube) were obtained, but methods are suggested to accomplish this. Instead, complete closure resulted in the cases described, with gratifying results to the patients. If there is complete tubal closure, one must create a permanent opening in the tympanic membrane.

HITSCHLER, Philadelphia.

CONFIRMATION OF NORMAL DISCRIMINATION LOSS FOR SPEECH ON C. I. D. AUDITORY TEST W-22. JOHN F. CORSO, Laryngoscope 67:365 (April) 1957.

One hundred thirty-nine normal but untrained subjects between the ages of 18 and 24 were given the C. I. D. Auditory Test W-22. The exact procedure is described. The obtained mean discrimination loss for speech was 2.32%. This was compared with the loss as determined for experienced and trained listeners, 1.1%.

It is gratifying that the two figures are in such close agreement.

HITSCHLER, Philadelphia.

ENDAURAL MODIFIED RADICAL MASTOIDECTOMY. JAMES A. MOORE. Laryngoscope 67:521 (June) 1957.

Various forms of the modified radical mastoid operation will suffice in the majority of cases of chronic ear suppuration. This is so because in most cases the disease is limited mainly to the mastoid antrum or attic or both. By exteriorizing the involved parts, the middle ear cavity can be left more or less intact, sealed off from the exteriorized cavity. In the Heath operation the antrum is exteriorized, in the Bondy, both the attic and the antrum. Cholesteatoma is not a contraindication, provided it is limited to the above-mentioned areas. The matrix is best left intact. Sometimes one or more ossicle has to be removed. If the cholesteatoma dips down into the tympanic cavity, it must be removed in toto and the lower tympanic cavity sealed off by a tympanoplastic procedure. It is emphasized that each case must be treated individually. The patient's safety must be guarded, the disease removed, and if possible, the hearing maintained or improved. Case reports with audiograms are included.

HITSCHLER, Philadelphia.

A STUDY OF 500 STAPES MOBILIZATIONS. VICTOR GOODHILL and ARTHUR L. HOLCOMB, Laryngoscope 67:615 (July) 1957.

This is a very good and complete discourse on the present status of mobilization of the stapes. It includes the reasons and theories as to why certain procedures will succeed or fail. The techniques are discussed in detail, and there is particular emphasis on the newer methods of footplate manipulations. It is to be noted that footplate fenestration in itself is condemned. In this, early closure is the rule. Also it is pointed out that the real purpose of the operation is an entire mobile ossicular chain.

Complications are not severe. The primary one at present is a utricular type of vertigo which follows footplate procedures, but this was of short duration.

Analysis of the results of 500 cases are given in detail. The precentage of successes rose as experience was gained, being significantly higher in the last 100 than in the first 100. The only preoperative prognostic indication was the air conduction. The better the air conduction, the better the prognosis.

HITSCHLER, Philadelphia.

INDICATIONS FOR MOBILIZATIONS OF THE STAPES. RICHARD J. BELLUCCI, New York J. Med. 57:3151 (Oct.) 1957.

Bellucci is connected with the Hearing and Speech Clinic and the Otology Laboratory in the Manhattan Eye, Ear and Throat Hospital. He states that aural surgery is now widely accepted for the relief of severe deafness. Furthermore, some 70% of "ideal candidates" have maintained a degree of improvment above the 20 db. level. One must now choose between fenestration and mobilization. "The percentage of failures is still high." Therefore a patient who has tried surgery without improvement, whichever method is used, is loath to "try again."

Fenestration is a major operation, performed under general anesthesia, with all the discomforts attending any like procedure. On the contrary, stapes release can well be performed under local anesthesia, requires no especial preliminary preparation, and can be safely performed on "poor surgical risk" cases. Mobilization is difficult for the surgeon because he cannot completely visualize the oval window and must, therefore, work by touch rather than by sight. Fenestration, however, according to lately improved techniques is well under the eye of the surgeon. One might assume that aged patients have a firmer fixation of the footplate than younger persons. This is theoretically true, but actual results are likely to be as good in each age level compared with another.

Before deciding on the "operation of choice," the surgeon should know his patient well, including, of course, a complete physical and mental spectrum. Logically, one might conclude that it is well to try mobilization first. If it fails, fenestration is then in order. It may be refused, according to the judgment of the patient, or accepted as a last recourse.

I. W. VOORHEES, New York.

SOME EXPERIENCES OF PLASTIC REPAIR OF THE DRUM MEMBRANE AND THE TYMPANUM. PAUL FRENCKNER, Acta oto-laryng. 45:455 (Sept.-Oct.) 1955.

The author reports experiences with 50 cases during the period 1951-1955. Two methods were used. One was a pedicle flap from the auditory canal. The other combined the pedicle flap with the tympanoplasty of Wullstein and Zollner. There were three types of cases: (1) dry perforations of the ear drum; (2) chronic otitis without cholesteatoma, and (3) chronic otitis with cholesteatoma. While the pedicle flap is technically more difficult, it gives a better drum membrane end-result. It is not to be recommended in the face of severe infection. Particularly in cases with cholesteatoma, two-stage surgery is indicated. First is a conservative type of radical mastoidectomy, with retention of maximum of canal skin and drum membrane; second, tympanoplasty. The purpose was to find out serviceability of different methods in cases of a variety of severity.

LEWY, Chicago.

STAPEDECTOMY AND AUTOTRANSPLANTATION OF OSSICLES. AGNAR HALL and CURT RYTZNER, Acta oto-laryng. 47:318 (April) 1957.

The authors present an interesting and brief report of a new surgical method for establishing action and continuity of sound transmission in the middle ear. This technique is used where interruption of the ossicular chain has occurred. The specific purpose is to restore and establish connection between the tympanic membrane and the oval window by autogenous transplantation of ossicles. This connection can be satisfactorily obtained by using either the malleus or the incus. The incus is preferable if available. One end of the transplanted ossicle is placed in the oval window, the other end is placed against the ear drum. When the operation is successful close to normal hearing is obtained. This corresponds to the single ossicle for sound transmission that exists in the bird's ear.

LEWY, Chicago.

POSTOPERATIVE RESULTS OF TYMPANOPLASTY IN THE COURSE OF THE RADICAL OPERATION. JAN SEKULA, Acta oto-laryng. 47:353 (April) 1947.

In doing tympanoplasty in the course of radical mastoidectomy the author found that using free skin grafts in the middle ear was an effective method of gaining marked improvement in hearing. However, experience with failures indicated that it was not always possible to create suitable nutrition for the flap. This, of course, indicates the need for further improvement in method and technique.

It seemed evident that good functioning of the new graft required a patent Eustachian tube. If there are no labyrinthine complications, no active bone inflammation, and if there is fairly efficient function of the middle ear, they feel that tympanoplasty should be applied in every surgical case of chronic otitis media.

LEWY, Chicago.

SODIUM BICARBONATE IN THE TREATMENT OF VERTIGO. JOSEPH FURSTNER, Ann. oto-laryng. 74:414 (June) 1957.

The author reports on 22 patients with Ménière's syndrome who were treated by intravenous injection of 50 cc. of a sterile 7% solution. Excellent results were obtained in those cases where the lesion was both reversible and peripheral. In six cases there was a complete control of the vertigo. In eight additional cases there was a marked improvement in that the attacks of vertigo and nausea were less frequent and of shorter duration. However, eight patients did not show any improvement.

Rotation and caloric stimulation of these patients before and after the intravenous injection showed marked changes in the responses. Nystagmus and vertigo were both depressed. While the effect of the sodium bicarbonate solution cannot be explained chemically, it is thought that it produces (1) a slowing of the action of the otoliths, and (2) a relaxation of the angiospasm. There may be a decrease in the viscosity of the blood serum and thus a change in the albumin-globulin ratio. Another theory is that there is a change in the electrolyte balance in the perilymph and endolymph.

In conclusion, the author believes that this procedure is a valuable adjunct to treatment especially in the reversible cases.

PERSKY, Philadelphia.

VASOMOTER INFLUENCES IN AUDITORY DISTURBANCES. M. HENRY ALOIN, J. franç. oto-rhino-laryng. 6:158 (March-April) 1957.

The author outlines the entire vasomotor supply to the ear, stressing effects of the entire vasomotor system and the vascular system. He indicates the various stimulants that may act both as constrictors and as dilators. He presents an extensive bibliography of a number of investigators with their theories as to the mechanism of these vasomotor reactions. These reactions may be either central or peripheral and may be stimulated by various drugs, psychic phenomena, hormonal reactions, and frank pathologic conditions, either centrally or peripheral. The author also describes the various vasomotor changes that may occur in influencing audition, tinnitus, and equilibrium. A great deal of stress has been placed on the role of allergy.

PERSKY, Philadelphia.

THE PRINCIPLES AND PRACTICE OF BONE-CONDUCTION AUDIOMETRY: A REVIEW OF THE PRESENT POSITION. J. D. HOOD, Proc. Roy. Soc. Med. 50:689 (Sept.) 1957.

Hood states that air audiometry is well understood and well applied, but "the basic principles of bone audiometry are . . . far from being generally understood." Consequently the tests are not well performed, and hence fail to provide vital diagnostic information. Hood is a researcher in the National Hospital, Queen Square, London.

The article is highly technical, and therefore not easily abstracted, but fundamental features of the tests procedure are as follows: 1. Establish the air-conduction audiogram of both ears, with masking if necessary of the untested ear. 2. Find the bone conduction threshold with the bone conductor applied to the mastoid of the tested ear without masking the untested ear. 3. Apply the masking noise of the appropriate band to the untested ear by means of an "insert receiver," and find a bone-conduction threshold reading. 4. Apply the "shadowing procedure" by increasing the level of the masking noise to 10 db. above threshold, and retest the bone-conduction threshold. "There is a 'changeover' point which gives the true bone-conduction threshold of the tested ear. . . . It is to be hoped that the methods described will find useful application in improving the design of standard audiometric equipment." There are 12 figures and 4 references to guide laboratory workers in this field . . . . Clinically, bone-conduction is of especial interest at this time because of the tiny pick-up receivers in the ends of broad-barred spectacles (glasses).

I. W. VOORHEES, New York.

RECENT WORK ON THE BIOCHEMISTRY OF THE LABYRINTHINE FLUIDS. L. CITRON and D. EXLEY, Proc. Roy. Soc. Med. 50:697 (Sept.) 1957.

Citron works in the Otological Research Unit in the National Hospital, Queen Square, London. Exley is engaged in the Cell Metabolism Unit, Department of Biochemistry, University of Oxford. This experimental work was undertaken to throw light on the chemistry of the labyrinthine fluid as possibly affecting such conditions as Ménière's disease, and the Scheibe type of hereditary cochleosaccular degeneration. . . . The subject matter is highly technical. Cats and guinea pigs were the aids to science. Methods used were (*a*) collection of fluid samples; (*b*) microchemical analysis. Chief conclusions were that utricular endolymph has a high potassium concentration and a low sodium concentration compared with perilymph or cerebrospinal fluid; also that the protein level in perilymph is higher than that of the other fluids. There is much in the tables and legends appended thereto regarding the chemical elements found. While conclusions seem to have little present clinical value, "It is hoped that further analytical work will bring us nearer to a fuller understanding of these mechanisms, and so to the rational treatment of Ménière's disease."

I. W. VOORHEES, New York.

THE HEREDITARY FACTOR IN DEAFNESS. MICHEL PORTMANN and JEAN-LOUIS DAVID, Rev. laryng. 78:205 (March-April) 1957.

In an analysis of 300 patients studied at the Phonoaudiology Center the authors seem to have been able to associate heredity more or less on a Mendelian basis. In many cases the dominant gene was quite apparent but in others this was questionable. From their observation they believe that deafness may be of an obvious hereditary character such as in otosclerosis and some of the dominant or recessive characters in perceptive deafness. Other types of deafness may seem to be acquired, but here, too, there is a hereditary element, such as allergy, neuroarthritis, and the tendency to rhinopharyngeal and tubotympanic infections. Finally, some types of deafness which appear to be acquired are present only because there is a hereditary tendency towards deafness and it can be influenced by exogenous toxins and occupational noises. The authors believe that hereditary factors either apparent or latent involves every patient with deafness and decries the fact that the otologist does not seem to give this problem the attention it deserves. If the hereditary deficiency can be found, prophylactic measures would prevent many subjects from becoming deaf.

PERSKY, Philadelphia.

DISPLACEMENT IRRIGATION WITH ANTIBIOTICS IN CHRONIC MIDDLE EAR INFECTIONS. A. LASKIEWICZ, Rev. laryng. 78:321 (May-June) 1957.

The author discusses 30 patients with chronic middle ear infection. He employs the displacement irrigation of the middle ear, using the various antibiotics, while some of these patients have been treated empirically by one or another antibiotic. Selectivity tests were made and specific treatment was given to a number of his patients. After an apparent cessation of the discharge the author has applied an artificial tympanic membrane made of cellophane to the perforation. In a few cases this not only improved the hearing acuity but decreased the tinnitus, and finally effected the closure of the perforation.

PERSKY, Philadelphia.

CANCER OF THE EAR. LACHAPELE and CHAUVERGNE, Rev. laryng. 78:341 (May-June) 1957.

The authors discuss the various types of cancer of the ear. Those involving (*a*) the external auditory canal and (*b*) the middle ear. The histology reveals the basal-cell epitheliomas, undifferentiated epitheliomas, intermediary epitheliomas, glandular epitheliomas, and lymphosarcoma.

Therapy was roentgen therapy, radium therapy, or surgery, either alone or in association with either radium or roentgen therapy. Only one-fifth of their cases showed a survival of more than five years. The authors again stress the importance of early diagnosis and the co-ordination of both the surgeon and the roentgenologist to complete the entire triad of treatment, namely surgery, roentgen therapy, and radium therapy.

PERSKY, Philadelphia.

Larynx

THE PRESENT PLACE OF THE LARYNGOFISSURE OPERATION. T. A. HARRIS, J. Laryng. & Otol. 71:249-256 (April) 1957.

The early history of the laryngofissure operation is briefly reviewed, and the many indications for the use of the operative procedure are discussed. It is the only manner in which impacted foreign bodies of the larynx which have resisted removal by direct laryngoscopy can be successfully removed. The author briefly discusses the staging of carcinomas of the larynx and goes on to say that laryngofissure has its greatest value when used to remove a malignant lesion from one vocal cord. The indications for such a procedure are discussed and the comparative value of surgical treatment versus irradiation is considered at length. The article ends with a summation of the advantages offered by a laryngofissure operation for malignant growths of the vocal cord over the results obtained with irradiation.

LEJEUNE and KEARBY, New Orleans.

TONGUE DEPRESSOR ASPIRATED INTO THE LARYNX. PHILIP M. FELDMAN, WALTER A. SCHMITZ and WALTER LENTINO, New York J. Med. 57:2562 (Aug.) 1957.

A 36-year-old man had been alcoholized for one week. During a convulsion, a well-intentioned nurse tried to insert a wooden tongue blade between his teeth to save biting his tongue. The distal end broke off and was aspirated. Symptoms were hoarseness, difficulty of inspiration, pain on swallowing, and a feeling of "something sticking" in his throat. The foreign body was located by x-ray at the level of the fifth cervical to the first thoracic vertebra. Under thorough cocainization, the foreign body was visualized by direct tube, which exposed the anterior commissure. The object was grasped by suitable forceps, and was removed with the withdrawing of the instruments.

The author's search of the literature shows that wood is rarely found in the respiratory tract, and there seems to be no report of a broken tongue depressor therein. A warning is in order not to try to force the "bite" of an unconscious person other than with a strong metal tool, if then.

ABSTRACTOR'S COMMENT: Biting one's tongue is characteristic in many cases of convulsions (epilepsy). The powerful masseters and adjacent muscles are capable of amputating the tip. To get the jaws to separate may call for watchful waiting, and the instantaneous insertion of a mouth gag which will not slip down the oropharynx into larynx or esophagus. This case history seems to prove that wood is the one worst thing to use in such an emergency.

I. W. VOORHEES, New York.

HEREDITY IN CANCER OF THE LARYNX. RENATO FIOR, Acta oto-laryng. 47:407 (May) 1957.

The paper summarizes a study of 170 patients with proved carcinoma of the larynx. It was shown that of relatives of 32 of them, 18.82% suffered from some form of malignant tumor. A comparison made with data of a matched control group showed a family incidence of only 9.41%. It is fairly well accepted that human families vary in their susceptibility to cancer. Under these circumstances the control of extrinsic factors might tend to reduce cancer incidence potentially present in high cancer incidence families.

LEWY, Chicago.

INDICATION FOR TRACHEOTOMY. P. MOUNIER KUHN, J. GAILLORD, H. CHASSEGROS, and P. MEREAUD. J. franç. oto-rhino-laryng. 6:103 (Jan.-Feb.) 1957.

The authors review tracheotomy and its problems, and in discussing the earlier indications they believe that a new evaluation should be made for this procedure. They have established the following indications:

1. Medical conditions such as (*a*) respiratory form of poliomyelitis; (*b*) the bulbar type; (*c*) tetanus; (*d*) coma due to medical conditions or various forms of intoxication, such as botulism or severe eclampsia; (*e*) marked respiratory insufficiencies.

2. Surgical conditions: (*a*) cervical facial surgery; (*b*) postoperative bronchopathies; (*c*) thoracic surgery, (*d*) severe traumatic injuries to the thorax; (*e*) cranial cerebral traumas which cause respiratory difficulties; (*f*) neurosurgical procedures.

PERSKY, Philadelphia.

RHABDOMYOSARCOMA OF THE LARYNX. PIAGET, COUDERT and JAUDEL DE GRENOBLE, J. franç. oto-rhino-laryng. 6:552 (July-Aug.) 1957.

The authors present a 76-year-old man who complained of progressive hoarseness for the past year. This has reached the state of marked dyspnea. A laryngeal examination was performed with great difficulty, and a large pedunculated mass was noted at the anterior end of the left vocal cord, but the cords appeared to be freely movable.

An emergency tracheotomy was performed. Subsequently, a thyrotomy was done, and a mass about the size of a date was removed from the left vocal cord. The entire area was electrocoagulated. The subsequent treatment consisted primarily of radiation, the patient receiving 8000 r to three areas.

The pathologic report was a rhabdomyosarcoma.

The patient made an uneventful convalescence, but seven months later there was a recurrence of the growth with ulceration and a marked dysphagia. This condition progressed rapidly to death.

The authors believe that this tumor acts like an epithelioma, and that treatment should be surgery plus radiotherapy.

PERSKY, Philadelphia.

Nose

PROBLEMS RELATING TO NASAL IMPLANTS AND GRAFTS. IRVING B. GOLDMAN, Eye, Ear, Nose & Throat Month. 35:798 (Dec.) 1956.

Careful evaluation of the results following the use of nasal implants and grafts is frequently difficult because of the failure of patients to return for follow-up examination. In an attempt at a long-term study of a series of 56 cancellous iliac implants in the nose, in the author's clinic, only two patients returned for examination. Statistics relating to the use of grafts in general are unreliable. Though cases may be reported in which certain types of implants appear effective, the use of the same material is frequently discontinued. This has been true of ivory, of bovine bone, and of polyethylene. Careful evaluation of the end results is essential, and this must be impartial.

Polyethylene is useful as a dorsal graft, provided the nasal tip is not corrected at the same time. The implants found exit in the author's cases through the primary intercartilaginous incisions. Autogenous cartilage grafts exhibit central necrosis of the graft and a tendency to warp. In some cases this may be due to a faulty preparation of the recipient area.

Autogenous bone grafts are more suitable, notwithstanding the fact that they are subject to occasional absorption. In certain cases, the grafts appear to "take" well, and the results appear satisfactory. Subtotal or complete absorption takes place in many patients, and this may occur very slowly. In two cases which the author reported previously, progressive absorption began five months after insertion and complete dissolution took place in seven months. It is the author's belief that the absorption rate is well over 50%. At this time, of long-term use of all varieties of nasal implants could be recorded and scientifically there is no ideal implant or graft. Goldman urges the establishment of a center where reports evaluated.

JENNES, Waterbury, Conn.

RHINOPLASTIC CONTINGENCIES. IRVING B. GOLDMAN, Eye, Ear, Nose & Throat Month. 36:49 (Jan.) 1957.

An interesting series of case reports is presented illustrating various forms of untoward incidents which may occur during a rhinoplasty. The patient may fail to provide the surgeon with certain information which might have contraindicated the operation. Surgery on patients with seasonal allergy can result in a profound reaction if surgery is performed during the season of allergic activity. A skin allergy to adhesive tape, abnormally thin and sensitive skin, blood dyscrasias, and drug idiosyncrasies to either the premedication or the anesthetic drug may be responsible for untoward occurrences during or after surgery.

Goldman cautions that at the first indications of an unexpected hazard, the operation should be stopped if it has not gone far, and if dangerous symptoms are manifested late in the course of the operation, the operation must be completed speedily. Despite the likelihood

of an unsatisfactory end-result, a "live patient" with an "imperfect nose" is better than a "moribund patient" with a perfect one.

JENNES, Waterbury, Conn.

RHINOPLASTIC TERMINOLOGY. IRVING B. GOLDMAN, Eye, Ear, Nose & Throat Month. 36:116 (Feb.); 176 (March) 1957.

Training of rhinoplastic surgeons has shown a striking advance in recent years. There is no substitute for the diligent application of the student in learning the technique of rhinoplastic surgery and in developing the esthetic concept which is so important if the cosmetic result is to match the physiologic improvement. Unfortunately, a surgeon having experienced poor results will seek to improve his results by utilizing or inventing new instruments. These will scarcely replace a knowledge of the operative steps, and of the limitations that exist in certain cases, and the ability that comes with experience to correctly evaluate one's work.

It is obvious that in rhinoplastic surgery postsurgical faults, even though minor, are conspicuous. It is impossible to hide one's bad results. It is important, therefore, to have a clear idea of what may be obtainable surgically and of explaining this carefully to the patient. The reliance upon "tricks" to obtain good results represents a faulty philosophy. A successful result in a difficult case may be due to a carefully executed procedure or maneuver which is incorrectly interpreted as a trick. It is time for an awareness that one becomes a good surgeon only by a sound understanding of the concepts of rhinoplastic surgery, and not by seeking a quick solution based on inadequate knowledge of the operative steps. Grasping at innovations is not the answer.

Nomenclature has been a stumbling block in the study of rhinoplasty. Several different and conflicting terms have been used to describe one portion of the nose. The nasal dorsum is described in the B. N. A. nomenclature as being situated in the ventral portion of the nose. The same region is also referred to as the anterior portion of the nose. This can, and does become confusing.

Goldman feels that it is extremely important that the B. N. A. terminology be accepted without dissent for the designation of position and direction of parts of the nose independent of body position. The terms cephalic, caudal, ventral, and dorsal may be applied to the nose, irrespective of the position of the patient. It is essential that terminology be uniform, and that it be accepted by all writing in this field.

JENNES, Waterbury, Conn.

A SIMPLIFIED TECHNIQUE FOR MAXILLARY SINUS IRRIGATION THROUGH THE NATURAL OSTIUM. JOHN W. GRAVES, Eye, Ear, Nose & Throat Month. 36:167 (March) 1957.

A modified natural ostium cannula has been used in more than 3600 patients with antral disease in the past 10 years without complication, and in only four patients was it impossible to enter the natural ostium. Differences in percentage of successful entrance through the natural ostium is dependent upon knowledge of anatomy and upon training and experience. Irrigation of the natural ostium is the treatment of choice and should be used in preferance to other methods.

JENNES, Waterbury, Conn.

CARCINOMA OF THE ANTRUM: AN ANALYSIS OF SIXTY CASES WITH SPECIAL REFERENCE TO PRIMARY SURGICAL EXTIRPATION. HAROLD G. TABB, Laryngoscope 67:269 (April) 1957.

Carcinoma of the antrum is a rare and usually fatal disease. The treatment of choice is a combination of surgery and radiation, but because the disease is so infrequently discovered early when it is localized, the results are poor. Of 60 cases collected in 1940-1955, only 7 are alive today, and only 4 of these have passed the five-year period. The ages range from 24 to 77 years, with twice as many men as women. Chronic irritation seems to play a part in etiology. The histologic form is mainly epidermoid or squamous-cell. The tumors spread by direct extension and by lymphatic metastasis. As the metastases are often found only at autopsy, the actual percentage rate is probably greater than usually suspected.

Symptoms are discussed in detail. Pain and swelling are listed as the earliest, but these are really in all probability, late symptoms. Before the sinus becomes filled with the tumor, other milder symptoms, such as unilateral discharge, blockage, and epistaxsis must surely

occur. Because of failure to realize the significance of such symptoms, carcinoma of the antrum is not discovered in its early stages. It is suggested that, if a so-called maxillary sinusitis does not respond to usual treatment within three to four weeks, a Caldwell-Luc approach be made and a biopsy be taken. Radical surgery—maxillectomy, plus irradiation—is indicated in most positive cases. The technique is discussed in detail. Hypotensive anesthesia is desirable because of bleeding. Other aspects discussed are pre- and postoperative care, indications for eye removal, management of metastases, and the cause of death. Emphasized are the gloomy aspects of the disease and the necessity of using desperate (radical) remedies.

HITSCHLER, Philadelphia.

News and Comment

ANNOUNCEMENTS

Course in Otologic Surgery.—A course on surgical technique, "The Surgery of Deafness," will be given under the direction of Prof. A. Aubin, Professor of Otorhinolaryngology at the Faculté de Médecine de Paris, June 9-14, 1958.

The course will cover the recent progress in otologic surgery, including procedures such as tympanomastoidectomy, fenestration, stapes mobilization, and tympanoplasty.

The tentative schedule is as follows:

8:00 a. m. to 12:30 p. m.—Operative Demonstrations by Drs. Aboulker, P. Andre, Prof. M. Aubry, F. Berard, J. Bouche, J. Bourdial, P. Clerc, H. Guillon, H. Henrot, Y. Lallemant, R. Maspetiol, M. Ombredanne, E. Poncet, J. Pinel, J. Salomon-Danic, and G. Senechal.

2:30 to 5:00 p. m.—Conferences followed by discussions—Prof. M. Sourdille and Drs. M. Bouchet, M. Lallemant, L. Leroux, J. Lemoyne, and A. Moulonguet, under the direction of Prof. M. Aubry, Drs. P. Aboulker, J. Bouche, P. Clerc, J.-J. Debain, and MM. J.-E. Fournier and A. Gribenski.

5:30 to 7:00 p. m.—Practical work, demonstrations, and exercises, presentation of instruments and motion pictures.

The enrollment is limited to 60.

For complete details write to Course de Technique Chirurgicale, Service d'Oto-Rhino-Laryngologie, Pavillon Isambert, Hôpital Lariboisière, 2, Rue Ambroise-Paré, Paris, 10.

American Otological Society.—The 91st annual meeting of the American Otological Society will be held at Hotel Fairmont, San Francisco, May 17-18, 1958.

Directory of Otolaryngological Societies *

FOREIGN AND INTERNATIONAL

Canadian Otolaryngological Society
(Société Canadienne d'Otolaryngologie)

President: Dr. Robert T. Hayes, 42 Cobourg St., St. John, N. B.
Secretary: Dr. Donald M. MacRae, 34 Spring Garden Rd., Halifax, N. S.
Place: Nova Scotian Hotel, Halifax, N. S. Time: June 9-11, 1958.

Venezuela Otorhinolaryngology Society

President: Dr. Alfredo Celis Pérez.
Secretary: Dr. Jesús Miralles.

International Association of Allergology

President: Dr. Samuel M. Feinberg, 303 E. Chicago Ave., Chicago 11.
Secretary-General: Dr. José Quintero Fossas, Paseo 313, Vedado, Havana, Cuba.
Meeting: Third International Congress of Allergology.
Place: Paris. Time: Oct. 19-26, 1958.

International Bronchoesophagological Society

President: Dr. Jo Ono, Tokyo.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Seventh International Congress of Bronchoesophagology.
Place: Kyoto, Japan. Time: Sept. 12-14, 1958.

International College of Surgeons, Section of Ophthalmology and Otolaryngology

President: Dr. E. G. Gill, 711 S. Jefferson St., Roanoke, Va.
Secretary-Treasurer: Dr. Louis Savitt, 3403 W. Lawrence Ave., Chicago 25.

International Congress of Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Leyden, Netherlands.

International Course in Paedo-Audiology

General Secretary: Dr. H. C. Huizing, 182 Oostersingel, Groningen, Netherlands.
Place: Groningen, Netherlands.

Pan-American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

President: Dr. José Gros, Havana, Cuba.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Meeting: Sixth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: Brazil. Time: Aug. 12, 1958.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Alden Miller, 500 S. Lucas Ave., Los Angeles.
Secretary: Dr. Hugh A. Kuhn, 112 Rimbach St., Hammond, Ind.
Place: San Francisco. Time: June 23-27, 1958.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Erling W. Hansen, 90 S. Ninth St., Minneapolis.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

American Board of Otolaryngology

President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse 10, N. Y.
Secretary-Treasurer: Dr. Dean M. Lierle, University Hospitals, Iowa City.

American Broncho-Esophagological Association

President: Dr. Walter Hoover, 605 Commonwealth Building, Boston.
Secretary: Dr. F. Johnson Putney, 1719 Rittenhouse Sq., Philadelphia 3.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

American Laryngological Association

President: Dr. Harry P. Schenk, 326 S. 19th St., Philadelphia 3.
Secretary: Dr. James H. Maxwell, University Hospital, Ann Arbor, Mich.
Place: Fairmont Hotel, San Francisco. Time: May 19-20, 1958.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. Lawrence R. Boies, University Hospital, Minneapolis 14.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Place: Mark Hopkins Hotel, San Francisco. Time: May 21-23, 1958.
Place: The Homestead, Hot Springs, Va. Time: March, 1959.

SECTIONS:

Eastern.—Chairman: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40.
Southern.—Chairman: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Middle.—Chairman: Dr. Francis L. Lederer, 860 N. Lake Shore Drive, Chicago.
Western.—Chairman: Dr. Ben. R. Dysart, 960 E. Green St., Pasadena 1, Calif.

American Rhinologic Society

President: Dr. Russell I. Williams, 408 Hynds Bldg., Cheyenne, Wyo.
Secretary: Dr. Robert M. Hansen, 1735 N. Wheeler Ave., Portland, Ore.
Annual Clinical Session: Illinois Masonic Hospital, Chicago, October, 1958.
Annual Meeting: Chicago, October, 1958.

American Otological Society, Inc.

President: Dr. Dean M. Lierle, State University of Iowa, Iowa City.
Secretary: Dr. Lawrence R. Boies, University Hospitals, Minneapolis 14.
Place: Fairmont Hotel, San Francisco. Time: May 17-18, 1958.
Place: The Homestead, Hot Springs, Va. Time: 1959.

American Otorhinologic Society for Plastic Surgery, Inc.

President: Dr. Armand L. Caron, 36 Pleasant St., Worcester, Mass.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16.

American Society of Facial Plastic Surgery

President: Dr. Irvin J. Fine, 505 New Brunswick Ave., Perth Amboy, N. J.
Secretary: Dr. Samuel M. Bloom, 123 E. 83rd St., New York 28.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. Joseph W. Hampsey, Grant Building, Pittsburgh 19.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6.
Place: Palmer House, Chicago. Time: Oct. 16-17, 1958.

Otosclerosis Study Group

President: Dr. Joseph A. Sullivan, 174 St. George St., Toronto 5, Canada.
Secretary-Treasurer: Dr. Arthur L. Juers, 611 Brown Building, Louisville.

Society of Military Otolaryngologists

President: Capt. William C. Livingood, U. S. N. (MC).
Secretary-Treasurer: Lt. Col. Stanley H. Bear, USAF(MC), 3810th USAF Hospital, Maxwell AFB, Alabama.

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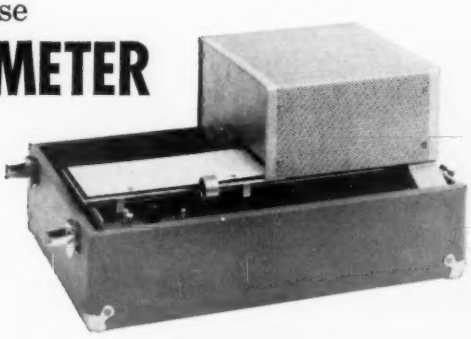

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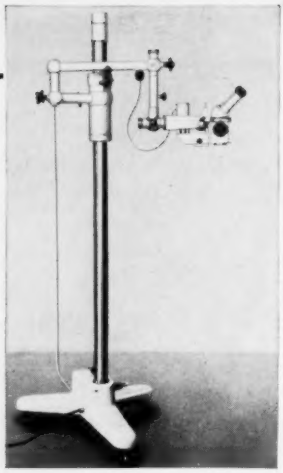

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Ref. Archives of Otolaryngology, February 1953

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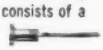

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